THE PATHOLOGY
OF
MEMORY

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CONTRIBUTORS

RAYMOND D. ADAMS
JACQUES BARBIZET
SAMUEL H. BARONDES
DIETRICH BLUMER
S. BRION
D. E. BROADBENT
JEROME S. BRUNER
ELISABETH CANY
CHARLES N. COFER
BÖRJE CRONHOLM
NORMAN GESCHWIND
H. HECAEN
ARTHUR KOESTLER
V. A. KRAL
LAWRENCE S. KUBIE
GEORGE A. MILLER
JOHN C. NEMIAH
MICHAEL I. POSNER
LEO POSTMAN
KARL H. PRIBRAM
GEORGE A. TALLAND
A. EARL WALKER
MOYRA WILLIAMS
O. L. ZANGWILL

THE PATHOLOGY OF MEMORY

Edited by

George A. Talland

and

Nancy C. Waugh

DEPARTMENT OF PSYCHIATRY
MASSACHUSETTS GENERAL HOSPITAL
BOSTON, MASSACHUSETTS

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LIST OF CONTRIBUTORS

Numbers in parentheses indicate the pages on which the authors' contributions begin.

RAYMOND D. ADAMS (91), Massachusetts General Hospital, Boston, Massachusetts

JACQUES BARBIZET (49), Faculté Médecin des Hôpitaux de Paris, Paris, France

SAMUEL H. BARONDES (117), Department of Psychiatry, Albert Einstein College of Medicine, Yeshiva University, Bronx, New York

DIETRICH BLUMER (65), The Johns Hopkins Hospital, Division of Neurological Surgery, Baltimore, Maryland

S. BRION (29), Laboratoire D'Anatomie Pathologique de la Clinique des Maladies Mentales, Paris, France

D. E. BROADBENT (167), Applied Psychology Research Unit, Cambridge, England

JEROME S. BRUNER (253), Harvard University, Center for Cognitive Studies, Cambridge, Massachusetts

ELISABETH CANY (49), Faculté Médecin des Hôpitaux de Paris, Paris, France

CHARLES N. COFER (211), Department of Psychology, The Pennsylvania State University, University Park, Pennsylvania

BÖRJE CRONHOLM (81), Psychiatriska Kliniken, Karolinska Sjukhuset, Stockholm, Sweden

NORMAN GESCHWIND (3), Veterans' Administration Hospital, Boston, Massachusetts

H. HECAEN (9), Centre Neurochirurgical, Hôpital Sainte-Anne, Paris, France

ARTHUR KOESTLER (261), 8, Montpelier Square, London, S.W. 7, England
V. A. KRAL (41), McGill University, Allan Memorial Institute, Montreal, Quebec, Canada
LAWRENCE S. KUBIE (237), The Johns Hopkins University School of Medicine, Baltimore, Maryland
GEORGE A. MILLER (223), The Rockefeller University, New York, New York
JOHN C. NEMIAH (107), Beth Israel Hospital, and Harvard Medical School, Boston, Massachusetts
MICHAEL I. POSNER (173), Department of Psychology, University of Oregon, Eugene, Oregon
LEO POSTMAN (195), University of California, Institute of Human Learning, Berkeley, California
KARL H. PRIBRAM (127), Stanford University School of Medicine, Stanford Medical Center, Palo Alto, California
GEORGE A. TALLAND° (273), Harvard Medical School, Boston, Massachusetts
A. EARL WALKER (65), The Johns Hopkins University School of Medicine, Baltimore, Maryland
MOYRA WILLIAMS (75), Department of Psychiatry, Addenbrooke's Hospital, Cambridge, England
O. L. ZANGWILL (161), The Psychological Laboratory, University of Cambridge, Cambridge, England

---

° Deceased.

PREFACE

The ability to remember both specific events and general rules is in many ways basic to every other higher mental process in man. It is therefore not surprising that over the past few years experimental scientists have turned in increasing numbers to the important questions of how we remember—and why, in the normal course of events, we forget. Very few of them, however, have made any attempt to study disorders of memory, even though it is almost axiomatic that one can often learn as much about a system by studying its failures as by describing its successes, and even though any general theory of memory will surely have to cover pathological processes as well as normal ones.

One purpose of the present volume, therefore, is to enable the academic scientist to become better acquainted with the variety of memory disorders commonly encountered by the clinician. He will see that many of them are characterized by a striking dissociation between various aspects of memory that are often difficult or perhaps even impossible to isolate precisely, or to observe independently of one another, in the normal human subject. The facts of abnormal forgetting should accordingly be of great potential use to the behavioral theorist: they may be able to provide new types of evidence in support of some models of memory while requiring the rejection or modification of others.

Another purpose of this volume is to acquaint the clinician with the academic scientist's current thoughts on how memory operates in general and on how its operation is best measured. Knowledge in this area has grown at a tremendous pace in recent years, so much so that it is possible to offer only a representative sample of present-day theories and experimental techniques. One would hope that the clinician might find this survey of help in his attempts to devise more sensitive and sophisticated means of evaluating the memory disorders that he encounters in his practice. It is important that such tests sample as wide a variety of functions as possible and that they be calibrated on large numbers of normal individuals, for only when we know what the average person is capable of remembering can we say precisely in what respect and to what extent memory has failed in a neurological patient.

Finally, this work may be of some special interest to the neurobiologist: in learning about the diverse nature of memory disorders and about the varieties of normal remembering in the human subject, he may evolve some new ideas as to just what it is that he should try to explain at the level of cellular behavior. It is probably safe to predict that his task will turn out to be at least as complicated as the mental processes that he will eventually want to account for.

I have grouped the contents of this book into four general categories. First, some descriptions of the nature and etiology of memory disorders in various clinical populations: that is to say, what it is that persons with various kinds of organic lesions or functional disorders can and cannot remember. Second, some observations on how learning and retention are affected when we deliberately tamper with certain biochemical and anatomical systems in infra-human subjects. Research of this nature can in many instances provide new hypotheses about the physiological basis of memory in the human being, and it can enable existing hypotheses to be tested directly. Third, some general ideas and specific facts about how and why the normal individual remembers and forgets. The last paper, by George A. Talland, deals with the relevance of academic research on memory to the description and assessment of memory disorders in man. It persuasively urges the need for more give-and-take between pure and applied behavioral science.

The various chapters are based on the proceedings of a symposium organized by George Talland, and held in October of 1967. His purpose was to get a diverse group of scientists to exchange ideas, not merely in the expectation that they would teach each other some new facts about memory but also, and more importantly, in the hope that they might thereby be led to formulate some new questions and develop some new points of view. George Talland died before these proceedings could be prepared for publication; he asked me shortly before his death if I would see the job through. I trust that the book will attest to the importance of his conviction that clinician and scholar each stand to gain a great deal by listening to what the other has to say; and I hope that it will in some measure reward the enthusiasm and skill of his efforts to being about a closer rapprochement between the two.

I am grateful to Drs. Pierre M. Dreyfus and David McNeill for help with some of the chapters that have been translated into English, and to Dr. Michael McGuire for his answers to a wide variety of questions. The symposium at which these papers were originally presented was made possible by a grant to Harvard Medical School from the National Institute of Mental Health, United States Public Health Service.

NANCY C. WAUGH

Boston, Massachusetts
June, 1969

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THE PATHOLOGY OF MEMORY

I

MEMORY DISORDERS IN CLINICAL POPULATIONS

THE APRAXIAS AND AGNOSIAS[1]

Norman Geschwind

The purpose of this discussion is to consider whether the apraxias and agnosias may be usefully considered to be disturbances of memory. It would be impossible to summarize here the clinical disorders subsumed under these rubrics or in any way to do justice to the extensive discussions and disagreements in the literature. Thus, this discussion is at best a diagrammatic introduction to a complex problem.

Although there had been occasional descriptions in the earlier literature of what we would now recognize as apraxia, it was H. Liepmann (1900) who first used this term in its modern sense and gave the first extensive clinical descriptions. We may define the apraxias, slightly modifying his description, as disturbances of learned purposeful movement not accounted for by weakness, sensory loss, reflex changes, alterations in tone or elementary muscular coordination, higher level reflex release, or general disturbance of comprehension, attention, or state of awareness. It is worth stressing that we are speaking of a loss of *learned* movements, and thus it is reasonable to inquire whether the apraxias are disorders of memory. The agnosias correspondingly may be defined as disturbances of identification via some sensory modality in the absence of changes in elementary sensory function, comprehension, alertness, or attention. We use both apraxia and agnosia in a narrow sense, so as to exclude certain syndromes of obscure mechanism such as frontal gait difficulty, problems in dressing, constructional difficulty, and disturbances in finger recognition.

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[1] From the Boston University Aphasia Research Center and the Boston Veterans Administration Hospital. Some of the work reported here was supported by Grant-NB 06209 from the National Institutes of Health to the Boston University School of Medicine.

The first problem that must be mentioned is whether any such disorders meeting these criteria do exist, since there have been certain authors who have asserted that all cases could be accounted for by more elementary disturbances. The answer must be unequivocally yes. Not only are there cases in the older literature, but also there are more recent cases. Thus the patient of Geschwind and Kaplan (1962) carried out commands correctly with the right hand but not with the left hand. The patient had a grasp reflex on the right, as well as some weakness and sensory loss, yet he carried out commands correctly with this hand. On the left side, which lacked all these elementary findings, commands were not carried out. Although we cannot give full details here of this patient who was surmised in life to be suffering from an infarction of the corpus callosum (subsequently confirmed at postmortem), the evidence is clear that the disturbances of movement of the left arm could be properly designated as apraxic by the definition given above.

In order to clarify the following discussion, let us specify the various meanings of the term "disturbances of memory." In one sense one can speak of a disturbance of storage, that is, the actual extirpation of a memory. In another sense one may speak of retrieval disturbances, where the memory is present but unavailable. A loss of storage implies that all stimuli will be ineffective in arousing the memory. A retrieval loss may be more specific: Certain stimuli may be ineffective while others continue to arouse the memory. It is obvious that it is much easier to disprove the presence of a storage disturbance than it is to prove the existence of such a disorder. If one can elicit the correct response by some stimulus, then one is dealing with a retrieval disorder. But if one fails to elicit the correct response, the possibility must always remain open that some other stimulus not thought of by the examiner might be effective.

It would be most tempting to assume that apraxias and agnosias could be the result of disturbances of storage, that the memory of how to do something or the memory of what a given visual stimulus is has simply been expunged. It appears, however, that this is probably not generally the case, as the following illustrations bring out.

Let us consider again the callosal patient of Geschwind and Kaplan mentioned earlier. He failed to carry out a verbal command with his left arm, for example, "Show me how you use a hammer," while he carried out such commands correctly with the right. The correct performance with the right hand excludes the possibility of a general storage loss. When given an actual hammer, the left hand would perform properly, thus showing that the right hemisphere also had stored the memory of the proper performance of the act. The left hand did not respond properly in response to a verbal stimulus. One could consider the disturbance of the left hand here as a specialized retrieval disturbance, a failure to perform in response to stimuli that come from the left hemisphere. The stored memory in the right hemisphere could be retrieved by means of stimuli directly reaching this hemisphere.

However, it is not always so easy to prove that the right hemisphere has a store of movements that can be retrieved only in special circumstances. Thus Liepmann and Maas (1907) pointed out that some patients with callosal lesions may fail to use the left hand properly not only on verbal command but also on imitation of the examiner or when given the actual object to handle. In these cases we have no evidence to suggest the presence of a right hemisphere store. Liepmann believed that the right hemisphere frequently might have no store of movements but simply might operate passively under the influence of memories stored in the left hemisphere. Unfortunately one cannot rule out the possibility that there is a right hemisphere store that has become inaccessible.

Parenthetically it may be seen from this that there are two different mechanisms that could account for right-handedness or, to put it more formally, for left hemisphere dominance for movement. One possibility is that the "memories for movement" are laid down in the left hemisphere and that the right hemisphere is run passively across the corpus callosum. In this instance "left-hemisphere dominance" is equivalent to "memory store laid down predominantly in left hemisphere." This was the mechanism that Liepmann accepted and which led him to say that the left hand is even clumsier than we think it is since most of the skill it possesses is borrowed—from the left hemisphere by way of the corpus callosum. The alternative mechanism is that the memories for movement are laid down substantially in both hemispheres but that retrieval is possible only from one. In this sense the left hemisphere is truly "dominant" over the right, which is not permitted to perform. Which of these mechanisms is correct, or whether varying admixtures of both occur, is a problem that awaits further clarification.

The apraxias so far mentioned have been only unilateral left apraxias, but if there is a left hemisphere store then it should be possible to produce a bilateral apraxia from a single left hemisphere lesion. I shall not go into detail on this point except to say that such cases are in fact observed. It is also of interest to note that while cases with unilateral *left* apraxia may be found readily, unilateral *right* apraxia is almost never seen. This too is compatible with the notion that the store in the left hemisphere generally supplies both hemispheres but that the right hemisphere must tap this store for movements of the left hand.

It thus appears that the apraxias may be divided into those which are disturbances of memory, involving either retrieval or storage, and those which are due to failure to transmit retrieved memories to the appropriate location.

A consideration of most cases of what have been called agnosias shows that these fall into two major groups. The first group is typified by Lissauer's (1889) classic original case of "mind-blindness." The patient whose spontaneous speech was normal failed to name objects presented to him visually, and indeed he also could not read or name colors. He could name objects presented to him tactilely. That this was not a general loss of the memories of what were visual objects was shown by the fact that his abnormalities of visual identification appeared only on the demand for naming. He behaved normally in his environment, for example, he knew what to do when presented with food and utensils. This patient at postmortem had an infarct of the left visual cortex as well as of the splenium of the corpus callosum. It seems clear that the patient could see only in the intact right visual cortex but could not transmit this visual information to the speech area. This could be regarded as a failure to retrieve verbal memories in response to a visual stimulus. I will not discuss here the problem as to why this lesion generally causes a more constricted syndrome consisting of alexia, frequently with color-naming disturbance, but with sparing of object-naming (Geschwind and Fusillo, 1966).

The other type of syndrome that has been called visual agnosia is quite different. I have had the opportunity to see three such cases, although none in great detail. They correspond to descriptions of similar cases in the literature. In this form the patient generally suffers from either a right posterior hemispheric lesion or a bilateral lesion. He has gross difficulty in visual identification of objects, but he can read. He can name the major classes of colors, but he fails on fine color distinctions. His visual disturbance is characteristic. He can identify small portions of objects, but he has great difficulty in putting these together. His failures are more striking with more complex visual arrays. Thus faces present a special problem for such patients. As yet we have no clear understanding of these disturbances, but they differ markedly from the first group described above. Are these truly loss of a store of the rules for integrating scattered visual information into the appropriate object? Or are they disturbances of perception (that is, not dependent on previous experience)?

In theory a general disturbance of visual memories is possible. Since we know that the major outflow from visual cortex is to association cortex, a bilateral destruction of visual association cortex should lead to a serious loss of the rules for identifying visual arrays. Unfortunately not enough of these patients have been studied to permit specification of the underlying physiology.

Let us return to the original problem of this paper, the conceptualization of the apraxias and agnosias as disorders of memory. Clearly, memory is involved in the laying down of the programs of motor activities and in the interpretation of what is perceived. Any simple notion that all these disorders are simply the result of the destruction of memory stores, however, must be rejected. Some of the syndromes are probably the result of damage to the stores; others are specialized failures of retrieval from an intact store; still others are the result of the destruction of outflow paths from an intact store. Only when these disorders are clearly distinguished will we be able to make an appropriate analysis of the role of memory loss in the higher disorders of movement and perception.

Summary

The terms "apraxia" and "agnosia" are used to cover disturbances in learned movement or in identification via a particular sensory modality that occur in the absence of a disturbance of either elementary motor or sensory function or of disorders of comprehension or awareness or which are disproportional to the defects in these areas. These disturbances cannot be regarded simply as the result of destruction of delimited cortical regions that are storehouses for memories of movement or sensory identification. Rather they comprise a group of separable syndromes resulting from a variety of physiological disruptions, some of which have been outlined in the above discussion and many of which remain to be discovered.

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CLINICO-ANATOMICAL AND NEUROLINGUISTIC ASPECTS OF APHASIA

H. Hecaen

By aphasia is meant disturbances in verbal communication due to circumscribed cerebral lesions. Such a definition is not particularly satisfactory, since the language disorder is defined by the focal lesion giving rise to it rather than by any special feature of the disorder itself.

The complex organization of language, of course, can be disordered at various levels of integration. So we must first specify which disturbances of verbal expression or reception we include in the term "aphasia." Although a clinician has no trouble in distinguishing a motor aphasia from a subcortical dysarthria, or a sensory aphasia from the verbal performance of an incoherent schizophrenic patient, the theoretical basis of such distinctions is often unclear. Very frequently criteria independent of the linguistic disturbance, for example, associated symptoms and cortical lesions, are taken into consideration in deciding whether and how to separate these disorders. A classic example of such a decision concerns the relation between intellectual disorders and sensory aphasia.

Linguistic methods and concepts seem useful in providing criteria for distinguishing between so-called aphasias and other disorders of language or speech. Aphasias thus may be considered as impairments of the formal language code at different levels of verbal performance. As far as emission is concerned, disturbances of the phonological system with the phonetic aspect preserved can distinguish expressive aphasias from paretic or ataxic disorders of speech and disturbances of the use of syntactic rules can differentiate receptive aphasias from demential semantic incoherency. In the field of receptive disorders we must distinguish between failures of phonemic discrimination (as in word deafness) and mere lowering of auditory acuity that may lead to poor detection of some of the sounds of language. At another level, failure to understand verbal commands must be distinguished from the incomprehension of dementia, which is not limited to the verbal code.

The delusional and schizophrenic patient's language, despite its neologisms, it agrammaticism, and its semantic anomalies, which resemble the jargon of Wernicke's aphasia, indicate not a disorganization of the code itself but rather the use of a *neocode* whose rules depend upon the underlying psychological disorder. General factors such as perseveration, disordered control, memory impairment, and disturbances of attention likewise may affect linguistic expression. They must be studied by the same methods in patients who present no aphasic disorders, that is, whose lesions are not known to give rise to any aphasic disorder.

Linguistics and psycholinguistics have clarified clinical observations and made definitions more rigorous. At first glance, however, they seem only to confirm the established findings of clinico-anatomical studies. Different taxonomic, distributional, and even generative and transformational linguistic models of analysis have proved very valuable for a better description of language disorders. But an important reservation must be made concerning the generative and transformational model. Chomsky (1967) holds that there is a difference between linguistic performance and linguistic competence. Although verbal performance presupposes competence (that is, the system of rules and procedures internalized by the subject, allowing for sound and meaning to be linked up in a certain order), performance makes use of much more information than that given by the associations of sound and meaning as determined by grammar. Performance operates under the constraints imposed by memory, time, and the organization of perceptual or productive strategy. Thus, according to Chomsky, we cannot use the rules of order and transformations that specify the grammar of human language to account for all the successive acts that intervene in the formation of sentences. Chomsky therefore is against applying generative and transformational models to the study of aphasia. This must not be forgotten—neither by ourselves nor by psycholinguists—when analyzing results of sentence generation and transformation tests.

One of the principles underlying our work has been not to consider the various different linguistic models as mutually contradictory. Each is used at a level defined by the verbal behavior under consideration, and the underlying physiopathological mechanisms can account very simply for the observed phenomena. For instance, the grammar of one sort of aphasia could well be defined by the characteristics of the model that describes it. We must add that the nature of the specific model used for each of the clinical groups permits a definition of the specific features of the grammar under consideration.

Analysis of the complex verbal behaviors observed in different syndromes shows that they can be dissociated and that under their apparent identity there are, in fact, concealed different mechanisms. Despite obvious pitfalls it is possible to go from models of function to models of dysfunction and even to take into account anatomic and physiological data as Jakobson (1964) and Luria (1964) have tried to do.

Moreover, with taxonomic and distributional models, quantitative linguistic methods can be used to study (1) the frequency distributions of parts of speech in different types of aphasia as compared with normal subjects (Wepman, Bock, Jones, & Van Pelt, 1956) as well as (2) the nature of the basic equations governing frequency of word usage in normal subjects (Howes, 1964). Improved techniques help make evaluation more objective. Progress in phonological description has been very valuable in the study of motor aphasia: Spectographic analysis seems to confirm that the disorder is on a phonological, and not on a phonetic, level.

In psycholinguistic research (see Oldfield, 1966) naming disorders are no longer studied merely statically, but rather the latency of the naming response is correlated with the frequency of use of the word and is compared to that observed in the normal subjects. Research upon the intelligibility of language as a function of signal-to-noise ratio should also be mentioned, even though there are very few such studies using clinical subjects. Communication and information theory have given rise to a number of experimental studies, but they have used primarily normal subjects.

Finally, studies of auditory hemispheric dominance via dichotic tests have shown that the left hemisphere is dominant in the perception of verbal sounds and the right in that of melodies. Recent work in this area (Shankweiler & Studdert-Kennedy, 1967) shows that left hemispheric dominance exists not only for the discrimination of meaningful units of language but also for phonemes and even their distinctive features.

We wish to integrate clinico-anatomical data that are over a century old with more recent results obtained via linguistic and psycholinguistic methods. By taking into account the relations of various language disorders to disorders of other semiotic systems, we hope to present a classification of the modalities of aphasia. We obviously do not regard it as definitive; we believe only that it reflects a stage in the development of research on language disorders. It can be useful only if it is considered as a clarification of the diverse data available, thus helping us to be more systematic in our observations and to carry out experimental research so as to test the validity of one or another classification.

Agreement between clinico-anatomical data and the preliminary results of linguistic analyses, although it may well be an artifact of our methods of investigation, encourages us to attempt a synthesis of this sort. It is already evident that multidisciplinary research seems to uphold the intuitive view of clinicians who insist upon the plurality of the forms of aphasia as against those who think of aphasia as a unitary phenomenon.

Complex syndromes first must be considered as cumulative effects, but they also must be examined as a means of studying interactions between the clinical phenomena that are found in isolation in so-called pure cases. From the analysis of correlations between the various disorders of verbal performance in a long series of observations randomly chosen, on the one hand, and the experimental analysis of as complete a sample of extreme clinical forms as is available, on the other hand, we may hope to discover whether the observed associations are necessary or merely fortuitous. Such a procedure must be complemented by the analysis of clinico-anatomical data and of the clinical concomitants of each type of pathological verbal behavior.

For the purposes of this classification, we have tried to take into account the form of the verbal behavior, emissive or receptive, and the channel wherein the disturbance appears; the structure of performance in spontaneous and elicited language as shown by linguistic analysis; and associations between clinical observations and the site of the lesion in the brain.

I. Classification of the Aphasias

A. Disorders of Spoken Language

1. *Expressive Aphasia*

This term refers to three types of aphasia. Two of them are very similar to each other, while the third is quite different.

a. Phonemic Aphasia. This is the classic motor aphasia, except that it is nearly always accompanied by writing disorders that seldom resemble the disturbance of spoken language either in nature or in degree. However serious the expressive disorder, verbal comprehension is rarely disturbed except in the very early stages of the disease; the understanding of written texts is always retained. Associated disorders are acalculia (written or mental); bucco-lingual-facial apraxia, especially when the speech disorder is acute; and sometimes left ideomotor apraxia. There is either a right hemiplegia or a brachio-facial or brachial monoplegia, at least initially; sensory disturbances are likewise found almost invariably. The disorder primarily affects oral and graphic expression.

Taxonomic analysis of spontaneous verbal production or of attempts at repetition by these subjects, provided the disorder does not limit speech to a few monosyllables, brings out the fact that the disturbance is at the phonemic level, such that the morphophonemic rules of the surface structures are conserved. Difficulties in production do not occur at the morphemic level but depend on the length or complexity of the item (monosyllables are pronounced better than polysyllables and nonsense syllables not as well as words of the same length).

A distributional analysis of these disorders suggests a hierarchy that takes into account phonemic subunits (distinctive features and articulatory components). There are certain quantitative differences in the distribution of these subunits: The vowels remain, whereas occlusives are pronounced relatively well; series of consonants are more troublesome than isolated consonants; and finally the frequency of consonant errors differs according to whether they are initial or terminal, the former being more frequent.

A study of the effect of sequential constraints shows that these disorders derive from the following phenomena: In the case of vowels whose formants modify the consonant transitions, the transformation is accentuated so that the consonant can be changed from one order into another; the phenomena of consonant, vowel, or even syllable anticipation show the substitution of a set of simpler or more frequent movements for less frequent or more complicated movements; and the phenomena of perseveration block movements already begun and induce reiteration at all levels.

It finally must be emphasized that the patients' production is very variable. All phonemes can be pronounced; however, certain defective distributions are more frequent than others. Thus the phonological system is conserved, as is confirmed by the fact that patients are capable of recognizing minimal pairs.

At the suprasegmental level, there is a slowing up of speech (prosody) that is variable and fairly selective, involving mainly the vowels (compensation phenomena), and the relative slurring of vowels (diphthongizing) that gives the French aphasic a foreign accent. The slowing up can be explained by the appearance of superfluous consonants or vowels.

As a general rule writing disorders are very serious except in pure motor aphasia, and their main characteristic is literal paragraphia; the grapheme generally can be executed, but at the word level there are omissions, distortions, additions, and substitutions of graphemes. The sentence level hardly ever is attained. Spelling, moreover, shows grave disorders. This agraphia of expressive aphasia is marked by three dissociations: The disorder affects only writing to dictation and spontaneous writing, while copying is unimpaired; the writing of dictated words is much better than that of nonsense syllables; and figures and numbers are generally well written.

This form of aphasia, anatomically speaking, corresponds classically to lesions at the base of left F3; but, in fact, it is often found with frontal posterior lesions. However, it is difficult to exclude lower Rolandic lesions, as has been shown by research with numerous posttraumatic cases (Conrad, 1954; Shiller, 1947; Russell & Espir, 1961) or cases of varied etiology (Hecaen & Angelergues, 1964) and as is suggested by the disorder's clinical concomitants.

b. Agrammatical Aphasia or Disorder of Syntactic Production. This variety of the preceding form is mainly distinguished by the absence of phonic disturbances in spontaneous production and by speech composed mainly of isolated words—nouns and verbs in the infinitive form—with few functional phrases. Again only oral and written language is disturbed. Clinical associations and the sites of lesions are the same as those found in motor aphasia.

The linguistic analysis begun by Pick (1913) and by Isserlin (1922) has been carried out systematically by Goodglass and Mayer (1958) and by Cohen and Hecaen (1965) via the use of a taxonomic model.

1. Agrammatism is defined as a deficit of primary articulation ac-

cording to A. Martinet or, in a different terminology, of the elementary parts of the immediate constituents. It therefore concerns intrapropositional syntactic relations.

2. Agrammatism is different from motor aphasia where the motor aphasic deficit affects the phonemic production programme since morphemes are emitted without any error in spontaneous speech, the phonic deficit appears only upon repetition of words and nonsense syllables. The specific distribution of errors is no longer at the initial, but rather at the final portion of the word. These two forms are distinguished by the difference between the patient's ability to handle words (easier for agrammatics) and nonsense syllables (easier for motor aphasics).

3. Difficulties in prosody are found in both motor aphasics and agrammatic aphasics. In both types of patient, speech is slowed down with a more or less controlled segmental deficit. In agrammatic patients, however, due to the difficulty they have in the concatenation of constituent elements, the utterance, of course, becomes reduced to sentences using only nouns and infinitives: Each of these elements nevertheless conserves a sentence value because of expressive intonation and accent.

The morphemic deficit is defined by a reduced syntactic pattern, a series of nominal utterances ordered and separated by a silence playing the part of a marker space, and conservation of lexemes (infinitive verbs included) with transformation capacity conserved (use of singular and plural forms, tenses, and so on). However, the poverty of the patient's lexical stock as compared with that of the normal subject's is likewise constant. In the test of "opposites," although morphological opposites cannot be given, lexical opposites, despite some deficit, are relatively easy for the patient to find. The general reduction of the sentence is shown by the substitution of implicit references for explicit references to the situation (suppression of *je*, *ici*, and *maintenant*) and by fewer syntactic markers. Writing disorders display the same features as disorders of spoken language with combinatory difficulties and a relatively intact vocabulary.

c. Conduction Aphasia or Phrastic Programming Disorder. The main features of this form are disturbances of repetition, of writing, and even of reading, which contrast with the absence of disorders of verbal reception.

Spontaneous language, despite some disturbance, shows that the speaker controls his verbal emission with frequent self-correction at all levels. Amnesic aphasia is often associated with conduction aphasia, but disorders of verbal recall are not essential to this form of aphasia. For instance, difficulty in finding opposites exists in conduction aphasia for morphological opposites but not for semantic opposites.

Conduction aphasia is associated with acalculia and disturbance of reproduction of rhythmical structures and frequently, despite some exceptions, with bilateral somatognosic and ideomotor or constructive apraxic disorders. There are no motor defects, sensory disorders are slight, and the visual field is generally unimpaired.

Anatomically speaking, lesions giving rise to this sort of aphasia are generally found in the posterior parietal area (angular and supramarginal gyri), perhaps associated with slight temporal lesions, in the posterior part of T1 and T2, in the second transverse gyrus, and in the temporal plane.

Linguistically speaking, conduction aphasia is an alteration of phrastic programming capacity in different kinds of verbal performance. Indeed, although the capacity to make sentences and to manipulate the grammatical code as a whole are unimpaired, the concatenation of elements is greatly disturbed, as is shown in tests of repetition and sentence generation.

In the repetition tests (nonsense syllables, words, and sentences), difficulties that are already evident in spontaneous speech become even more obvious. The longer the word, the greater the difficulty. There are always errors with utterances of more than four syllables, but the phonic elements remain similar to those of the model. The phonic difficulty is mainly of the syllabic reiteration type that stops the patient from finishing the phonic program of the word. Repetition difficulty depends on the length of the item presented, whether it be a word or nonsense syllable. The meaning of words does not help the repetition of more than three syllables; from here on, the length of the utterance is more important than its meaning. In sentence repetition the difficulty depends both on the number of words in the sentence and on the transformations it contains, that is, on its complexity.

In generating sentences from single words, these patients have great difficulty in putting three or four words into a simple sentence. In the rare sentences that are correctly generated, the pauses are abnormally long between syntagmatic groups, and paraphonias with self-correction appear within these units. These phonic difficulties are qualitatively similar to the phonemic difficulties or motor aphasics. Nevertheless, they have certain features that are different—for example, their appearance in the middle of a word and not at the beginning, the frequency of syllable iteration, and less difficulty with words of few syllables than with nonsense words of the same length. In conduction aphasia there are difficulties on the morphemic constituent level, while in motor aphasia these difficulties are on the production (praxic) level. Disturbances likewise occur that depend on the quantity of information in the concatenation of the constituents of the morpheme and of the sentence.

Writing disorders are constantly found. Graphic production shows auto control with frequent crossing out, overwriting, and stopping. The graphemes are correctly formed, but there are disturbances of the structure of words similar to the literal paraphasia in motor aphasia but with some telescoping or even substitutions or omission of lexemes. The writing of dictated single words is better than that of nonsense syllables. At the sentence level there is usually some impairment; subordinate and coordinate clauses may be formed, but at the same time function words are left out and there also are slight syntactic deficiencies. Copying remains unimpaired and figures and numbers are correctly written, but spelling is always faulty. Besides difficulties in reading out loud, there also are disturbances in comprehending complex written orders and in understanding a text.

To summarize, the deficit in conduction aphasia is found at the level of the morpheme, the syntagm, and the sentence; and it does not depend upon the channel used.

2. *Amnesic Aphasia*

Disturbance in word recall is part of sensory aphasia, but it may be so pronounced as to deserve separate consideration when other disturbances of language are hardly noticeable. In this case speech is marked by a failure to find words, with continual stopping and replacing of words and phrases by such words as (in French) "*machin*" or "*chose*" or an explanatory gesture. An object-naming test clearly shows up this deficit. The patient cannot find the name of the object and describes it by its use.

Pure clinical examples of this disorder are hard to find, since it is so often associated with other forms of aphasia, especially at the onset. In fact, it seems to assume several forms. Anatomical data confirm both the multiplicity of lesional sites and the importance of temporal lesions.

Linguistically speaking, the disturbance in choosing root-morphemes has two aspects:

1. In spontaneous speech, the morphemes that depend least on syntax, those which bear the least syntagmatic constraint, are the most likely to disappear: for instance, proper names whose meanings vary with the referential situation, and words that do not refer to a segment of speech, that is, names of objects.
2. In the naming test, where terms are out of context and thus func-

tion poorly as names, their disappearance is a function both of the familiarity of the object and of the frequency of use of the word.

3. *Sensory Aphasia*

Sensory aphasia comprises a specific set of disorders in the reception of verbal signs associated with disturbances in emission, recall, and control, together with disorders in writing and even of reading. Communication may fail completely because of the degree of paraphasia and the disturbance in recall.

Sensory aphasia may be associated with symptoms such as apraxia, acalculia, and finger agnosia. Except for acalculia, however, these symptoms are not constantly present and seem to depend upon the extent of the lesion that has occasioned the aphasia. The same holds for intellectual deterioration. The general neurological examination is negative, on the whole, except for a frequent impairment of the visual field. Lesions causing this disorder are found in the language area defined by Wernicke (1874) as the posterior part of T1 and extended by Dejerine (1914) to the posterior part of T2.

While this may be the general outline of sensory aphasia, the disorder subsumes a number of features which, in actual cases, are present in differing proportions. Some subjects seem to suffer from a specific form of the disorder. It is possible, therefore, to distinguish between three basic factors that in various combinations account for the more complex cases. These three factors may be denoted as verbal deafness, understanding disorders, and disorganization of attention.

Verbal deafness is the patient's incapacity to interpret verbal auditory stimuli, although his peripheral auditory capacity is not sufficiently impaired to account for the difficulty. Of course, it is a well-known fact that this disorder may appear in a pure form, that is, without disorders of emission, recall, reading, or writing (except for writing to dictation). In other cases, the picture is the following. First, the subject is totally unable to repeat words, nonsense syllables, or sentences: His attempted reproduction seems to be a mere string of syllables whose structure deviates farther from that of the original the longer the utterance. Spontaneous language retains a thematic coherence, although paraphasia and substitution make it hard to understand. Reading is much better than is the reception of verbal signs, but paralexia becomes more obvious with the growing complexity of a text. Copying is correct, but writing to dictation is impossible. Spontaneous writing is also impaired, but thematic coherence is preserved and paragraphias are relatively slight.

When verbal understanding is primarily impaired, the picture is quite different. The reception of verbal signs is preserved and words are repeated fairly easily when they have some meaning, as is shown by repetition tests; moreover, mispronounced words are accurately reproduced. The repetition of short sentences is correct, but orders given orally are very poorly executed and little help is afforded by the repetition of an order. Spontaneous language is very erratic: Its main feature is that the patient continually starts sentences without finishing them. Prosody is normal; there are few neological paraphasias and a great deal of agrammaticism; writing to dictation is more or less correct, but in spontaneous writing there are frequent paragraphias.

The third factor in sensory aphasia is a disorganization of attention. This factor has a specific effect on the linguistic behavior of patients. Since there is only very mild disorder of understanding or verbal deafness, the repetition of words, nonsense syllables, or sentences presents no difficulty. This disorder is very pronounced in spontaneous language and in grammatical transformation tests, with echolalia and distractibility as the two principal features.

The distractibility is apparent in the patient's inability to finish a sentence that he has started. This gives rise to successive changes of the meaning of what he has already said. The sentences pronounced are grammatically correct on the whole, but as soon as the patient goes beyond the kernel sentence, grammatical faults become frequent (for example, errors in agreement or disturbances in interpropositional relations). Echolalia manifests itself either in repetition of the examiner's speech or in perseveration within the patient's own speech.

There are few verified records of the lesions associated with these three kinds of sensory aphasia. In those that are available, it appears that the lesions are always in the left temporal lobe for all three types (lesions of the first temporal gyrus near the gyri of Heschl, posteroinferior temporal lesions, lesions of the temporal tip).

B. Disorders of Written Language

As we have seen, disorders in written language, at least when they concern encoding, are almost invariably associated with oral language disorders. However, the two are very frequently of different sorts. The features peculiar to the written as compared to the spoken code (perceptual and motor features, conditions of acquisition, and structural differences between the two codes) account for the partial autonomy of the former. Pathology has revealed certain discrepancies between disturbances in written and in oral language. These disorders may even be completely separated; indeed, it is even possible to find a dissociation between disorders in the reception and the production of written language.

For agraphias we shall make the following distinction: (1) agraphia in expressive aphasias and reception aphasias and (2) apraxic agraphias. Though there may be apraxic agraphia without oral language disturbance, these rare cases depend upon one of two mechanisms. First, the incapacity to produce graphemes is part either of an ideomotor disturbance or of a constructional disturbance. The subject in either case draws no better than he writes. However, there can be dissociation. Constructional apraxia in fact may be complete while graphemes can still be produced and vice versa, even when figures to be drawn are complex. Cases in which the spelling disorders are only slight show that the motor programming disorder may affect only writing. Apraxic agraphias, frequently associated with reading difficulties and finger agnosia and always with acalculia, are caused by left parietal lesions.

In the second type of apraxic agraphia without oral language disorder, the graphic disorder affects mainly the production of graphemes and the praxic disorder is limited to a left unilateral ideomotor apraxia. The latter would seem to depend upon psychomotor reactions of the grasp reflex or avoiding-reaction type (Denny-Brown, 1957). It should be mentioned that this disorder cannot be merely apraxic, since spelling and writing with alphabet blocks are also impaired. The causative lesion may be found in the left medial frontal region.

In the term "pure agraphias" are included all writing disorders not accompanied by impairment of the oral code, of reading, or of praxic activity. Such cases are very rare and some investigators doubt their very existence. Most published descriptions are ambiguous. Nevertheless, the author has encountered four cases of pure agraphia. The graphic disturbance, which is revealed both in writing with alphabet blocks and in spelling, is of the same form as disturbances found in expressive aphasia, but of course there is no phonemic disturbance. Associated clinical disorders also are similar. Clinical, electroencephalographic, and surgical data in observations tend to show left frontal lesions. We must here recall Exner's (1881) hypothesis about a center of graphic verbal images at the base of F2.

The main point is that besides these "pure" agraphias there are cases where the graphic disorder is far more severe than the oral one, although the latter is also present; thus there is a continuum running from cases of motor aphasia combined with graphic disorders of similar severity to cases of complete dissociation. Pure agraphia is merely a rare instance of this dissociation of speech and writing disorders and shows that the written code is extensively independent of the oral code.

C. THE ALEXIAS

Reading difficulties in Wernicke's aphasia merely externalize a general disorder of language comprehension. However, a distinction can be drawn between a general disorder with respect to written language (accompanied by a minimal disturbance with respect to oral language), on the one hand, and an isolated disorder of reading, on the other hand.

1. *The Alexia-Agraphia Syndrome*

Disturbances in graphic decoding and encoding are associated in this syndrome without necessarily being exactly parallel. On the whole one may say that letter recognition is unimpaired when there is a disturbance at the word level, the context seeming to help in the understanding of sentences and ordered phrases. Spaces are frequently ignored. The way words are read seems to show that the normal perceptual strategy used in reading is lost (Haslerud & Clark, 1957). The agraphia is similar to the reading difficulty in that there is a disintegration of the structure of words and a loss of the phonic-graphic correspondence. But apraxia may make reproduction of actual graphemes difficult. Copying is always impaired in the same way as is spontaneous writing or writing to dictation (mistakes in the formation of graphemes and morphemes, and whatever the syntactic constraints). Spelling also always is impaired, and the reading and writing of figures is often similarly disturbed.

One of the most frequent concomitant symptoms is an oral language disorder, but on the whole it is slight and hardly impairs the reception of verbal signs. The other concomitant symptoms are finger agnosia, apraxia (mainly constructional), and acalculia, the latter being the only invariably associated deficit. The lesional origin of this syndrome seems to be in the left parieto-temporo-occipital junction (supramarginal and angular gyri).

2. *Pure Alexia*

In contrast to pure agraphia, the existence of pure alexia is generally accepted; however, some writers think that it should be separated from language disorders and considered as a special optic agnosia or as the result of a more basic disorder (loss of the ability to structure Gestalten).

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Pure alexia differs from other reading disorders of cortical origin in that it is accompanied by no language disorder (fluency of speech and understanding are unimpaired), except for verbal recall; there are no spelling disorders and no impairment of the perceptual strategies used in reading; writing is normal—or, at least, the degrees of disturbance in reading and in dictated or spontaneous writing are quite different. Copying, on the other hand, is always impaired. Acalculia is frequent but usually slight; visual agnosia (colors and pictures) is variable. Homonymous right lateral hemianopia is always present. The lesions responsible may be in any area of the occipital lobe and may be the only cortical lesions found.

Analysis of the reading disorders found in occipital lesions leads us to believe that the several features that have been known for a long time, but considered merely as variations in the severity of the disorder, are in fact manifestations of three independent basic disturbances.

a. Verbal Alexia. When letter recognition and the spelling out of words are conserved, the difficulty increases with word length. Although simple letter sequences generally can be read, the reading of a complicated sequence or of a text fails, probably because of the effort of finding out what each letter is and linking it to the next one. Perhaps there also is some deficit of immediate memory so that a word read arduously is at best poorly retained. Writing is hardly impaired, and copying, although always impaired, is nevertheless not a slavish imitation of the model copied. Figures and even numbers can be read. Visual agnosia is a frequently associated symptom.

b. Literal Alexia. Except in rare cases the identification of letters remains possible, but errors are extremely numerous. Words are read better, especially lexemes; but function words and nonsense syllables cannot be identified. Paralexias, approximations based on the graphic form of the word and especially on its ending, are frequent, and even more remarkable is the observation that a word in the same semantic category as the word to be read is sometimes given and sometimes, on a second attempt, the patient gets nearer to the target. There is a great deal of approximation in the execution of written orders or in giving the gist of a text that has been read. Figure and number reading is more severely impaired than in verbal alexia.

c. Global Alexia. In this case no letter can be read, but spontaneous writing or writing to dictation is almost normal except for some spatial difficulties. It is here that kinesthetic compensation in visual reading disturbances seems to be the most frequent. Figures are usually recognized, but the loss of ability to read musical notation that has been found in several cases must be mentioned.

II. Sources of Individual Differences

When a systematic presentation of facts is made, as in the previous section, factors that may blur the edges of the various isolated forms necessarily must be omitted. Besides anatomical factors (nature of the lesion, its acute or chronic character, and mass effects) that considerably adulterate the purity of the syndrome, others also should be borne in mind: the subject's age, uncertain lateralization of function, sociocultural level, and structural factors concerned with language usage.

A. AGE

We will not discuss aphasia in children, other than to stress its relative rarity, the predominance of expressive disorders, and the relatively low incidence of receptive disturbances, whatever the site of the lesion and the fact that children tend to recover. Finally, one should note that aphasia occurs in right-handed children with lesions of the right hemisphere much more frequently than in adults.

B. FUNCTIONAL HEMISPHERIC LATERALIZATION

It is known that a right cerebral dominance for language functions is not the rule in left-handed subjects. Some authors even hold that left cerebral dominance is quasi-universal in these patients. Such a statement is too extreme, but aphasia certainly appears more often with left- than with right-sided lesions in left-handed patients. The reverse is generally true.

Although left-sided lesions often produce language disorders in left-handed patients (22 out of 37 cases in the series of Hecaen and de Ajuriaguerra, 1963), one should stress that the disorders are less severe than in right-handed patients and that recovery is quicker and more nearly complete. Furthermore, the language disorders present peculiar features: high frequency of articulatory disorders, often of an episodic nature with rare and mild disturbances of comprehension, vs moderate frequency of disorders in verbal recall and reading. Disorders of writing are also frequent, and apart from disturbances of the actual graphic code there is often a spatial dysgraphia, similar to that seen in right-handed patients with lesions in the minor hemisphere.

Right hemisphere lesions in left-handed subjects do not often produce aphasias. When present, they are usually of an episodic nature or are transitory, and they almost always have an expressive character; the only marked case of aphasia that we have observed was an amnesic aphasia.

A combined study of praxic and gnostic disorders, in this same group of left-handed patients with unilateral hemispheric lesions and in a group of right-handed patients, shows that mixed syndromes often occur in left-handed patients after lesions of either hemisphere. By mixed syndromes we mean disorders containing components of the usual syndromes seen with both right and left hemispheric lesions in right-handed patients. This study also appears to indicate that the hemispheric localization of the motor and sensory mechanisms underlying language is less consistent in left- than in right-handed subjects.

Cerebral bilaterality therefore is an important factor; this conclusion has been confirmed by the work of Milner, Branch, and Rasmussen (1964), who used the Wada test. It also seems reasonable to conclude that the areas subserving language modalities are widely distributed over one hemisphere in left-handed subjects. It therefore is evident that although there may be a left hemispheric dominance in left-handed patients, the variable organization of cortical representations make it impossible to clearly define the neurolinguistic aspects mentioned above.

C. Socio-Cultural Factor

Although this factor can be enormously important (for example, in illiteracy), its influence was found to be relatively small when the performance of a group of patients was compared with that of a control group, and it did not appear to affect the nature of the various disorders.

D. General Factors

The general factors consist of perseverance, deterioration, and disturbances of attentional vigilance, which have marked effects on linguistic performance. In order to study these factors the same series of linguistic tests was given to patients whose lesions had not produced aphasia (Parkinsonians and right hemisphere lesions) and to another group that displayed extensive general deterioration (senile and presenile dementias). The presence or absence of aphasia in the latter group made it possible to evaluate the extent to which either disorder interferes with language. By thus distinguishing general factors affecting verbal performance, we thought we could learn to recognize their effects on aphasic syndromes, without confusing them with true linguistic disturbances.

E. MEMORY FACTORS

The work of Luria, Sokolov, and Klimkowski (1967), among others, suggests that acoustic-amnesia aphasias may be linked to one sensory modality and even to some particular function; moreover, they may be produced by variously localized lesions. On the other hand, the study made of spontaneous and elicited language in cases of Korsakoff's syndrome did not reveal any disturbance in linguistic performance.

F. STRUCTURAL FACTORS

Two sorts of studies may be pursued to investigate the relation between the linguistic systems used by patients and their language disorders. The first study aims at establishing a comparative typology of linguistic deficits in the patient's native language. Preliminary results appear to indicate that despite differences in normal linguistic forms, the nature of the deficit does not vary.

It nevertheless is certain that such a study would be revealing if carried out on aphasics whose language differs fundamentally from the Indo-European varieties—either in the oral system (tone languages) or perhaps in the written code. Certain Japanese authors have shown the effects on reading and writing of different well-defined cerebral lesions. Such lesions affect Kanji (Chinese characters) and Kana (phonetic writing) in different ways. Analogous differences also occur in agraphia and alexia in the patients studied, that is, differential difficulty in the writing of words and nonsense syllables in motor aphasia or the dissociation between verbal alexia and literal alexia in reading disorders. Studies of Japanese aphasics have shed some light on these paradoxical discrepancies.

The second approach is to study language deficits in polyglots. Here there is a single language disorder, the effects of which may be analyzed concurrently in the different languages the patient has mastered previous to his illness. The study should be carried out on both the synchronic and on the diachronic level, taking not only structural factors into account but also the modes of acquisition of the language and the psychological and social factors.

On the synchronic plane, the work of Lambert and Fillembaum (1959) and of L'Hermitte, Hecaen, Dubois, Culioli, and Tabouret-Keller (1966) showed that whatever type of bilingualism is present (coordinated or composite, according to Osgood's terminology), the nature of the disorganization is similar in all languages used by the patient.

On the diachronic plane, analysis of the course of recovery has shown that despite the role of environmental influences the rule of Pitres (a good many exceptions to which have already been found) does not hold; the course of recovery is the same in the various languages used by a bilingual aphasic. Whether Pitres' rule does or does not apply, it is the patient's mastery of the language prior to his illness that determines his linguistic behavior. Factors of language usage stressed by Goldstein (1948) in fact do show quantitative variations, but the qualitative aspect never changes.

III. Conclusions

This chapter is an attempt to present a typology of the aphasias that includes both clinical and linguistic aspects. To this end, each type of verbal communication disorder has been placed in its proper context.

Thus aphasia of phonemic production is placed in the context of motor disorders, whereas conduction aphasia, despite its essentially expressive character, does not imply any motor disorder. On the contrary, its association signs indicate a disruption in the programming or categorizing of various other functional activities: arithmetic, somatognosia, praxias, gnosias, and disturbances of rhythmic and temporal organization.

With sensory aphasias the problem becomes more complex because they can appear in various contexts—they are sometimes rich in praxic and gnostic symptoms, while at other times they are unimportant or absent and only disorders of calculation are present. A more precise study of the structure of the language disorder, carried out in association with a clinical investigation, made it possible to bring out arguments in favor of the isolation of certain aspects of Wernicke's aphasia. Such an isolation is not justifiable on purely anatomical grounds. Amnesic aphasia also is seen in a wide variety of clinical contexts. Here again the different concomitants of the naming disturbance justify the investigation of features specific to the clinical syndrome within which they occur. For each type of syndrome described the topography of the lesion which seemed to be responsible has been given. Certainly the preceding accounts are schematic, but despite the efforts of authors who put forward global theories of cerebral function, this author tends to consider these accounts simply as a method of approach becoming more and more popular. Even Goldstein (1948), by using the concept of instrumentalities of language, lays stress on such systematization.

There obviously is a wide chasm separating the results of behavioral experiments on animals and language disorders in man. However, even pending their final interpretation, experiments such as those performed by Neff (1961) suggest the important effect of certain localized cortical lesions. Total destruction of all auditory projection areas, in cat or monkey, produced only one kind of hearing deficit; an inability to discriminate between different auditory stimulus patterns in temporal succession. These results become significant if it is assumed that verbal deafness is an inability to decode linguistic units into their correct temporal sequences. Experiments on animals also open up the possibility of linking, up to a point, neurophysiological events and behavioral phenomena.

It also would seem useful to bear in mind the results of the work of Hubel and Wiesel (1962, 1965), even though it deals with vision. Other experimental results (Katzuki, 1961) suggest that similar processes operate in the auditory system. The more complex information is derived from the more elementary and is transformed and retransformed at different functional levels. New information is extracted at each level and is integrated with previously acquired information. Thus, by analogy, one can conceive of a decoding of language undergoing successive integration at different functional levels, starting with an elementary auditory signal and ending with a significant unit.

Neurophysiology also teaches us (again mainly from studies of the visual system) that there is convergence onto one cell of information from many sensory modalities and also of nonspecific information. On the basis of such observations we can begin to understand how nonauditory activities can influence language, in much the same way that feedback mechanisms and their suppression might explain a patient's unawareness of his aphasia. Finally, the associative zones, which receive no primary projections, may provide a point of convergence for diverse sensory signals.

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KORSAKOFF'S SYNDROME: Clinico-Anatomical and Physiopathological Considerations

S. Brion

Korsakoff's syndrome is a clinical entity defined by four symptoms: first and most important, an amnesia for recent events, which includes an anterograde amnesia and a partial retrograde amnesia, although memory for remote events and didactically learned facts remains intact; second, a spatial or more commonly a temporal disorientation; third, some degree of confabulation; and last, occasional instances of false recognition.

On the basis of its etiology, *Korsakoff's disease*, or psychopolyneuritis of nutritional origin (in which the polyneuritis may be present in varying degrees) for a long time was distinguished from *Korsakoff's syndrome*, a psychological disorder that never includes polyneuritis and stems from various nonnutritional etiologies. In point of fact, the amnesia for recent events in these two classic categories is identical, and therefore it seems eminently reasonable to assume that there is a single Korsakoff's syndrome with various etiologies, of which the nutritional is the most common.

The present study is based on a sample of 40 patients who were observed in the author's clinic and who also were subjected to anatomical examination whenever feasible. The composition of the sample was as follows.

There were 20 alcoholic Korsakoff patients, of whom 12 were available for anatomical study; two nonalcoholic nutritional cases, who were studied only clinically; nine tumor cases, of whom seven were available for anatomical study; two vascular cases studied anatomically; two postencephalitic cases studied clinically; one case of anoxia studied anatomically; and four traumatic cases studied clinically. To these should be added 14 cases of presbyophrenia, including 11 with degenerative or mixed senile dementia, two with Pick's disease and one with Jakob-Creutzfeldt's disease.

The clinical, psychometric, and anatomical data that were derived from these patients shall be discussed and compared with certain other data described in the literature; then conclusions will be drawn from this analysis with respect to the pathology of the disease. Clinically speaking, the four most important features for discussion are mental confusion, confabulation, fixation, and progressive partial regression of the syndrome.

Korsakoff's syndrome and *mental confusion* are not the same thing, even though they have been equated with one another, principally by French writers. The two are in fact frequently associated in the initial acute phase of Korsakoff's syndrome; but then the mental confusion gradually clears up, independently of the other symptoms.

Confabulation is a consistent finding in Korsakoff's syndrome, but it is always secondary to a loss of memory for recent events, which is the principal characteristic of the disease. Confabulation can be categorized as follows, according to the degree to which it is present: (1) simple confabulation, which is poorly worked out and must often be induced, but which is always present, (2) confabulation involving recollections that may be true or false, which is commonly observed in the acute stage of Korsakoff's syndrome but gradually clears up and leaves behind only a residual amount of simple confabulation, and (3) confabulation involving purely imaginary events, often of a fantastic or grandiose nature, which is reminiscent of the delirium of the paraphrenic. The latter is rare and is seen mostly in posttraumatic cases. It is of short duration and perhaps depends on an associated mental confusion.

Confabulation is always part of the syndrome, and it is always better demonstrated by clinical interviews than by psychometric tests, since confabulation must be very prominent in order to be observable in the latter. The degree of confabulation perhaps is related to the location of the underlying lesion: It seems to be milder with temporal lobe lesions than with lesions of the cingulum. Unfortunately, however, we have very little anatomical information on posttraumatic cases, in which confabulation is often very pronounced.

Even though the term "amnesia of fixation" is in common usage, *fixation* (in the sense of immediate memory) remains intact in Korsakoff's syndrome; and this fact, previously demonstrated by Korsakoff himself, has been amply demonstrated by clinical and psychometric studies.

The *partial regression* of certain Korsakoff syndromes is of interest because it always follows the same pattern: First confabulation clears up gradually; then there is a progressive reduction of the retrograde amnesia and a re-arousal of previously inaccessible memory traces, at first fragmentary and then relatively coherent. The patient, however, will never remember completely the events that occurred during the acute period of his illness, and it is impossible to decide whether this fact reflects a defect in memory or simply the mental confusion that is found in most cases.

With respect to psychometric testing, Korsakoff's syndrome of alcoholic etiology is associated with an atypical learning curve, with frequent perseveration on the Benton test and on certain tests of the Wechsler memory scale and with certain anomalies in the reconstruction of figures in the Benton test, indicative of a sensory-motor deficit that is minor and is to be contrasted with the absence of Piotrowsky's organic psychosyndrome. The Rorschach test invariably brings out kinesthesias and perhaps confabulation as well (when the latter is clinically very rich). These results may be compared to those reported by Talland (1965), who has shown that a minor sensory deficit always exists in Korsakoff's syndrome of alcoholic origin.

The posttraumatic cases of Korsakoff's syndrome are frequently associated with a consistent organic psychosyndrome, which probably results from the diffusion of the critical cortical lesions. The postencephalitic and anoxic cases fall somewhere between these two extremes, according to the extent of the underlying cortical lesions.

Miss de Schonen (1967) has recently completed an experimental study of memory as a function of lesion localization, using as subjects tumor cases with diverse lesions and also Korsakoff cases of various etiologies (alcoholic, traumatic, and tumor). (The study was carried out in the neurosurgical service of Dr. Guiot at the Foch Medical and Surgical Center.) By presenting memoranda of varying degrees of meaningfulness and requiring either immediate or delayed recall, she was able to demonstrate significant variations in memory according to the anatomical site of the subjects' lesions. Thus, cases with unilateral temporal tumors display an atypical but monotonic learning curve, whereas the temporal course of learning by patients with unilateral frontal tumors is highly erratic. The parietal cases, wherever their lesions, experienced only slight difficulty even with minimally organized material; while the occipital cases were found to be free of any learning deficit when compared to the control group.

Moreover, although the span of immediate memory (as tested in the absence of any interfering activity) is normal in patients with Korsakoff's syndrome whatever its etiology, in the study first described, their short-term memory was found to be extremely poor for supraspan material at the two lowest levels of meaningfulness employed therein and mediocre for material at the two highest levels; their delayed recall of material at every level of difficulty was extremely poor. These various results seem to agree very well with the anatomical facts of Korsakoff's syndrome, which, as we shall see, require the presence of a very precisely localized bilateral lesion.

The clinico-anatomical facts of Korsakoff's syndrome are based in part on patients whose lesions have been verified at autopsy and in part on neurosurgical patients whose lesions have seldom been so verified. Anatomical data from cases of diverse etiologies follow.

Nutritional disease is encountered most frequently and is typically but not invariably associated with alcoholism. The classic lesions of Wernicke's encephalopathy are evident. The following three facts are particularly important.

1. A well-defined bilateral lesion in the mammillary bodies has appeared in all our cases. Two of our patients also displayed some bilateral softening of the body of the fornix, with secondary demyelinization at the junction of the fornix and the mammillary bodies. No similar lesion was found in any of the other cases.
2. The cortex is usually intact.
3. Frequent lesions in the area of the thalamus have been reported by Adams, Collins, and Victor (1962). They affect not only the midline structures but also the dorso-medial, pulvinar, and dorso-lateral nuclei, and they consist of an appreciable loss of neurons and of a diffuse and dense astrocytal gliosis. We have found similar lesions in all of our patients except four, in whom we found only mammillary lesions. Adams, Collins, and Victor nevertheless attribute more significance to the thalamic than to the bilateral mammillary lesions, and they cite in support of their argument the case of a patient with bilateral mammillary lesions who nevertheless presented no psychic disorder. Unfortunately, this case has not been described in detail in the technical literature.

Tumors responsible for Korsakoff's syndrome are usually bilateral and deeply set, whether located in the septal or frontal region of the cingulum or inside the third ventricle or at its base. These tumors affect the fornix, the mammillary bodies, and the anterior nuclei of the thalamus. The most frequent of these tumors is the craniopharingioma of the adult, which is represented in two-thirds of these cases.

Three facts are worth noting:

1. Korsakoff's syndrome of tumoral etiology is very rare in children, even though craniopharyngiomas are much more frequent in children than in adults.

2. Unilateral tumors very seldom cause Korsakoff's syndrome, except in rare cases of deep-seated neoplasms that may destroy part of the limbic system and compress the other—for example, as in one of our published cases. Unilateral hemispheric tumors may be erroneously blamed for Korsakoff's syndrome, on the other hand, either when mental confusion is mistaken for that syndrome or when there is the joint occurrence of a cerebral tumor and of Wernicke's encephalopathy caused by a secondary nutritional deficit.

3. Finally, intracranial hypertension seems to play an insignificant role in the etiology of Korsakoff's syndrome: Severe cases give rise to mental confusion instead.

Korsakoff's syndrome of *vascular etiology* is very rare but of great interest because its localization is in principle very precise, but one must bear in mind that the lesions are often multiple.

Certain forms are symmetrical and can be classified according to their locus in one of the following ways:

1. The double occipital softening as described by Dide and Botcazo (1902) and more recently by Boudin, Barbizet, Derouesné, and Van Amerongen (1967) involves the fimbria and gives rise to a secondary degeneration of the fornix and the mammillary bodies.

2. The bifrontal form as described by Mabille and Pitres (1913) and Zacher (1901) is characterized by a bilateral subcingular infarction that cuts off afferent input to the cingulate gyrus.

3. The bitemporal form, which may be a special instance of the bioccipital form or may in fact involve both of Ammon's horns, encompasses the cases described by Glees and Griffith (1952), by Victor, Angevine, Mancall, and Fisher (1961), and by Nomura and Ishikawa (1961). Infarcts here destroy both Ammon's horns as well as the fimbriae, occasionally in an asymmetrical manner as in the case of Victor, Angevine, Mancall, and Fisher, which showed a large infarct in the left temporal region with a lesser one in the right at the junction of the fimbria and the fornix.

4. The asymmetrical forms are even more exceptional and are limited to four cases, of which one has been observed by us and the other by Boudin (unpublished).

Our case demonstrated a visual agnosia, a right lateral homonymous hemianopia, and a Korsakoff's syndrome. Pathologically, the case demonstrated infarction of the mesial inferior temporo-occipital region and of the fimbria, secondary atrophy of the fornix and mammillary body, and three softenings situated in the lateral nucleus of the thalamus on the left. On the right side a small softening transected the middle portion of the bundle of Vicq d'Azyr within the thalamus.

The case of Boudin, which was even more asymmetrical, showed a right lateral homonymous hemianopia of long standing and a more recently acquired Korsakoff's syndrome associated with signs of frontal lobe affliction. The pathological anatomy revealed an old infarct in the left mesial inferior occipito-temporal region, infarction of the fimbria, and secondary atrophy of the fornix and mammillary body. On the right, an anterior cerebral infarction had destroyed the cingulate gyrus and half of the corpus callosum.

One should note that all of these vascular lesions are more or less asymmetrical, are bilateral, and are situated in the circuit of Papez. Korsakoff's syndrome caused by a unilateral lesion of this circuit is unkown. The only exception to this rule might be a case reported by Hegglin (1953). Here there was no softening but rather a unilateral ischemic lesion of Ammon's horn that had occurred in a nutritionally depleted patient whose mammillary system had not been examined.

The *postencephalitic forms* are most probably bitemporal, but only the cases of Conrad and Ule (1951) and that of Storring, Hauss, and Ule (1962) support this point. In these cases the frequent forgetting of didactic knowledge, which is rarely found in other instances of Korsakoff's syndrome, perhaps can be explained by the presence of more or less extensive temporal lesions. Half of the reported cases suffered from epileptic seizures, a fact that could account for the onset of memory disturbances.

There now exist in fact *pure postictal forms*, judging from a single, anatomically confirmed personal observation of a Korsakoff's syndrome that followed the onset of seizures caused by intensive adrenocortical hormone therapy for intractable asthma. In this case the temporal lesion was of the pure anoxic type, bilateral, symmetrical, and limited to Sommer's sector, with probably transynaptic degeneration of the left mammillary body and of the two anterior and two dorsolateral nuclei of the thalamus.

In the syndromes of *posttraumatic* origin, the lesions are probably diffuse and, because of a paucity of anatomical verifications, they are ill-defined.

Presbyophrenia, which is characterized clinically by the association of a Korsakoff's syndrome and intellectual deterioration, is usually associated with degenerative senile dementia, and the lesions, although diffuse, have a predilection for the fifth temporal convolution, Ammon's horn, and the mammillary bodies.

One also should mention the rare occurrence of presbyophrenia—in Pick's disease, in which severe lesions typified by a dense glial scar involve Ammon's horn. These lesions tend to be much more moderate in the usual form of the disease, where memory disturbances are absent.

One should further mention the thalamic form of Jakob-Creutzfeldt's disease, where the amnesic symptoms are massive and where symmetrical lesions destroy the anterior and dorso-lateral nuclei of the thalamus and to a lesser degree the dorso-medial nucleus.

Only the following *neurosurgical* cases will be cited:

1. There are the *mesial bitemporal lobectomies* of Scoville and Milner (1957) and the unilateral temporal ablations of Penfield and Milner (1958), in which cases there most likely exists a contralateral lesion analogous to the one seen in our postictal case. On the other hand, a so-called Korsakoff's syndrome caused by a unilateral temporal lobectomy has never been proved anatomically.
2. There are the *bilateral dorsomedial thalamic coagulations* of Spiegel, Wycis, Orchinik, and Freed (1956), which can be accompanied by Korsakoff's syndrome. Its variable and always transient nature leads one to believe that the lesion involves neighboring structures: the anterior or dorso-lateral nuclei of the thalamus or the tract of Vicq d'Azyr. The relative infrequency of Korsakoff's syndrome in these patients suggest that the dorso-medial nucleus plays an insignificant role in the etiology of Korsakoff's syndrome.
3. There are the *partial bilateral cingulectomies* of Whitty and Lewin (1960), which engender a transient Korsakoff's syndrome characterized by rich confabulation, although it occurs very rarely after medial bitemporal ablations.
4. Finally, an isolated case of Hassler and Riechert (1957), in which both fornices had been coagulated successfully, is the only case of this surgical series pathologically proved to have caused Korsakoff's syndrome. The procedure was followed by death in ten days as a result of associated lesions in the anterior hypothalamus.

Conclusions

From the preceding facts, a number of conclusions may be drawn concerning the nature of the behavioral deficit under discussion, its anatomical basis, and the organization of memory in general.

Regarding the nature of the symptoms of Korsakoff's syndrome, two *negative features* are generally agreed upon: (1) integrity of alertness, except for a mental confusion frequently observed during the acute phase, and (2) integrity of "fixation" in the sense of immediate or short-term memory. The equivocal term "amnesia of fixation" thereby may be discarded.

Two *positive features* characterize Korsakoff's syndrome: a disturbance in retention and a certain amount of confabulation.

The *defective retention of personal experiences* represents an alteration of long-term or integrative memory. There exists no satisfactory explanation for this deficit, although a temporary selective amnesia (Kleist, 1934; Van der Horst, 1956) and a defect in mental synthesis both have been suggested.

The latter hypothesis is more generally accepted. It either must assume a global defect to explain the apathy of the patients and accordingly consider the memory deficit to be a sequel of this defect or must assume a selective defect in the delayed retention of recent events, the apathy being secondary to or contingent upon the memory disturbance.

Confabulation is almost always present but may be limited to induced confabulatory responses. It is definitely secondary to the memory deficit, but the exact mechanism that generates it is unkown.

The *anatomical substrate* of Korsakoff's syndrome is now well known as a result of comparisons drawn between pathological cases of varied etiologies. A bilateral lesion involving the hippocampo-mammilo-thalamo-cingular limbic circuit appears to be responsible for the various manifestation of this syndrome. In fact, verified lesions involving the destruction of one side only never have been accompanied by Korsakoff's syndrome; conversely, bilateral lesions of the circuit, even though small or very asymmetrical, do engender memory deficits, provided they are not masked by an additional mental confusion caused by a spreading of the lesions. Uncertainty concerning some details does persist, however: If the role of Ammon's horn or the mammillary bodies is obvious, that of the cingulum is still poorly defined and that of the fornix is widely debated. Certain authors (Dott, 1938; Cairns & Mosberg, 1951) support the view that a lesion in the latter structure does not lead to a memory deficit. Furthermore, the role of the lateral dorsal nucleus of the thalamus must be determined more precisely, and perhaps it should be related to the limbic circuit. A final reservation results from the case of Adams *et al.* (1962), where a bilateral mammillary lesion apparently did not lead to psychic difficulties. Despite these few reservations it seems that the limbic system plays an important role, albeit a poorly specified one, in the genesis of Korsakoff's syndrome and consequently in the operation of normal long-term memory.

The clinico-anatomical observations described above have suggested two general notions regarding the organization of memory. The first notion is *functional* and refers to the existence of two successive stages of memory. These are (a) very *short-term* memory or immediate memory (occasionally called *fixation*), which functions normally in Korsakoff's syndrome, and (b) *long-term* or *integrative* memory, that is, the retention of experiences by means of a complex and as yet poorly understood mechanism of storage and recall, which in turn depends on an intermediary and as yet an equally unclear process of *coding*. The operation of long-term memory is selectively affected in Korsakoff's syndrome.

The second notion is *anatomical*: It rests upon our knowledge of the limbic system, in which a symmetrical or asymmetrical lesion (always bilateral) engenders a Korsakoff's syndrome, and the unilateral integrity of which is therefore essential, it would seem, to normal long-term retention.

A number of points in the physiopathological account of Korsakoff's syndrome remain obscure, however.

It is impossible to specify whether the mechanism afflicted in long-term memory is one of storage or one of retrieval, since the two processes are always clinically undissociable. It may be the case, moreover, that they depend on some common mechanism, such as coding, an assumption that would fit in well with Talland's (1965) reports of similar distortions in memory observed in the same patient after an interval of several months.

The exact role of the limbic circuit is unkown. It could act in a specific manner by temporarily storing memories in the course of consolidation, but it is more probable that its mode of action, that is, simply creating favorable conditions for storage and recall, is nonspecific. This notion could explain the apathy, the difficulty in rapid adaptation, and the stereotyped behavior of patients with Korsakoff's syndrome. It also would be in accord with results of animal studies conducted by Douglas and Pribram (1966), who believe that the limbic circuit functions in a nonspecific fashion as the "evaluator" of mistakes during learning.

The selective and predominant affliction of memory for recent events in Korsakoff cases and their excellent retention of remote events is still poorly understood. Attempts have been made to explain this difference on the basis of different sites of storage of the two types of memory, storage taking place initially in the limbic circuit and later becoming neocortical. It indeed is very likely that memory changes its form with time, by means as yet obscure.

Finally, the consistent retrograde character of the amnesia remains the stumbling block of many theories concerning Korsakoff's syndrome. The fact that it is retrograde indicates that immediate memory is not at fault, since the loss is of memories whose initial fixation may have been perfect. This fact tends to suggest, in other words, that Korsakoff's syndrome is not a simple defect of storage (which would explain an anterograde amnesia but not its retrograde counterpart). It has been known, however, since the work of Albert (1966a,b) that while the consolidation of memories is not instantaneous the time it requires can be measured experimentally in hours rather than in months, years, or decades. An extensive retrograde amnesia therefore cannot be explained on the basis of defective consolidation of recent memories.

One ends up thinking that retrograde amnesia might be best understood as a deficit in recall. Only in this way, perhaps, can one explain failures in the resurrection of memories that have been stored in a normal fashion. The possibility of an isolated deficit of recall seems to have been demonstrated by Albert in experiments that result in temporary blockage of recall without the actual destruction of memory traces.

Finally, it is possible that alteration of the limbic system results in deficient coding and thus not only in a disorder of storage, which may result in anterograde amnesia, but also in a disorder of retrieval, which would explain the consistent retrograde amnestic component of Korsakoff's syndrome.

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MEMORY DISORDERS IN OLD AGE AND SENILITY

V. A. Kral

The following presentation is a report on studies of memory disorders of aged people, including both well-preserved subjects and those suffering from psychoses of the senium. The sample comprised more than 1000 subjects and was taken from four different groups. The first consisted of well-preserved old people without clinically manifest psychiatric or neurological symptomatology who were living in the community or were residents of an old people's home. The second was made up of well- preserved old people who came to see us because they felt that their memory was failing and became afraid of future deterioration. The third group comprised aged people who were admitted to hospitals because of senile or arteriosclerotic brain disease. The fourth group was formed of chronologically old patients who were admitted to the hospital because of mental disorders of various kinds, such as depressions, schizophrenia, and organic conditions other than psychoses of the senium.

The investigation comprised psychiatric, neurological, psychological, and laboratory examinations. The observation period ranged from 2 to 12 years.

Although the incidence of memory disorders in the aged is not known, mainly because of the lack of representative epidemiological studies, clinical observation shows that a decline in memory function commonly occurs and is significantly correlated with advancing age. Psychometric tests demonstrate a loss of memory scores in all aging individuals as compared with young adults (Jones & Kaplan, 1956; Jerome, 1959). In one of the studies a group of 52 well-preserved old people with an average age of 78.4 years achieved a mean memory score of 83.7 out of a possible 117 achieved by young adults, demonstrating a loss of approximately 28% (Kral & Wigdor, 1963). Another study showed that in a group of 162 residents of an old people's home, who were 60 years old or more, the percentage of subjects with memory loss increased from decade to decade, whereas the percentage of subjects with clinically preserved memory function decreased correspondingly. Moreover, the subjects in the older decades had lower memory scores than those in the younger ones (Kral, 1958). There was, however, a remarkably wide range in scores achieved by the individual subjects within the decennial subgroups. This led to the assumption that qualitatively different types of memory impairment might be hidden behind the quantitative differences in memory scores, which actually was found to be the case. Two types of memory impairment could be demonstrated in all subgroups, from the youngest of 60–70 years to the oldest of 90 years and over, both of them significantly related to increasing age (Kral, 1959).

The first type is characterized by the inability of the subjects to recall certain data and parts of an experience, like a name, a place, or a date, whereas the experience as such can be recalled. The same data that are not available for recollection on one occasion can be recalled at another time. Furthermore, the forgotten data belong to the remote rather than to the recent past. This type of memory dysfunction occurs with approximately equal frequency in both sexes and progresses relatively slowly. There was no significant difference as to survival time and death rate between the subjects with this type of forgetfulness and the subjects without significant memory impairment.

The second type of senescent memory impairment occurs significantly more frequently in women than in men and is characterized by the inability of the subject to recall events of the recent past, whereby not only relatively unimportant data and parts of an experience but the experience as such cannot be recalled. Further observation showed that the subjects with this type of forgetfulness became disoriented and some also developed confabulations which, however, tended to disappear with progressive deterioration. On psychological testing the subjects with the second type show significantly lower memory scores; they have a significantly higher death rate and a shorter survival time than the subjects with the first type (Wigdor & Kral, 1961).

This type of senescent forgetfulness is essentially indentical with the senile amnestic syndrome.

Out of 60 residents of an old people's home with a mean age of about 80 years, without clinically manifest memory deficit or with only the first type of senescent forgetfulness, 19 (31.6%) died within a 4-year observation period, whereas in the same period of time 21 out of 34 subjects of the same age with the second type, that is, 61.7%, died. This difference in the death rates was statistically highly significant.

Table I

Incidence of Death within a 4-Year Observation Period in an Old People's Home

Memory function	Number of subjects	Number of deaths	Percentage of deaths
Normal or "benign" dysfunction	60	19	31.7
Malignant dysfunction	34	21	61.7
Total	94	40	42.6

During the same observation period only one subject, a woman with the first type of memory dysfunction, developed a senile amnestic syndrome that subsequently led to her institutionalization, whereas all surviving residents with the second type had to be placed in a closed psychiatric ward.

It was for these reasons that Kral suggested the terms "benign" and "malignant," respectively, for the types of senescent forgetfulness found in more or less well-preserved old people (Kral, 1962).

The data, as reported so far, tend to show that the type of memory dysfunction present in the aged can be taken as an indicator of the general health of the individual: the "benign" type as an expression of a "senium naturale," the "malignant" type—the senile amnestic syndrome—as one of a senium ex morbo.

This view was substantiated by more extensive studies on the aged population of a mental hospital, whereby the functional status of 696 patients, with a mean age of 75.3 years, was assessed at regular intervals over a period of 3 years. It was found that 45% of the patients with an amnestic syndrome were dead at the end of the 3-year observation period, while of the patients of the same age with functional psychoses, but without memory deficit or with only the "benign" type of senescent forgetfulness, 21% were dead by that time. This difference was statistically highly significant. Moreover, in the patients with organic disease who were still living at the end of the 3-year observation period, there was a very significant decline in the assessment scores of the following three variables: memory function, sphincter control, and general activity. No significant decline in these assessment categories occurred during the same observation period in the patients of the same age with functional psychoses (Kral, Cahn, & Mueller, 1964).

At the end of a 5-year survey of the same group, 312 out of the total sample material of 696 patients, that is, 45%, were dead. The death rate for the organic psychoses with an amnestic syndrome was 57% (196 out of 343), while that for the functional psychoses was 29% (84 out of 290). This difference also was statistically highly significant. Within the organic group the mortality was considerably higher in the first part of the observation period than in the second part. Out of the 196 patients deceased after 5 years, 154 had died within the first 3 years, most of them within the first year of the observation period.

Among the functional parameters investigated, impaired memory function of the "malignant" type was the one most significantly correlated with shortened survival time and increased death rate. The other parameters—reduced activity and sphincter control, which together with memory impairment formed the ominous prognostic triad in our 3-year survey—still held an important place, but only secondary to the parameter of memory impairment (Kral & Mueller, 1966).

No significant correlation was found between specific causes of death and the diagnostic groups. Senile marasmus, arteriosclerotic heart disease leading to coronary thrombosis and/or cardiac failure, pneumonia, cerebral hemorrhage, and infarction were the immediate causes of death. Their distribution was fairly even between the main diagnostic groups: organic vs functional, with the exception of cerebrovascular accidents that were found less frequently in seniles than in the arteriosclerotics (Kral, 1964). In other words, the senile amnestic syndrome is not related to any specific cause of death; it rather seems to be an expression of an increased liability to die from the same causes that are responsible for the death of other subjects of the same age group. This would indicate that the underlying pathological process, the senile brain disease, either by its nature or because of its localization or by a combination of these factors, has rendered the individual so affected even less resistant to random stresses than the aging process per se would warrant.

It is at the present stage of our knowledge not possible to formulate a precise opinion on the relationship between the amnestic syndrome and the diminished resistance of the senile psychotic patient. However, on the basis of what we know to date, a hypothetical formulation can be made.

Table II
Incidence of Death within a 5-Year Observation Period – Mental Hospital Material

Diagnostic group	Dead within 3 years	Percentage	Dead within 5 years	Percentage	Alive after 5 years	Percentage	Total
Organic psychoses	154	45	196	57	147	43	343
Functional psychoses	60	21	84	29	206	71	290
Miscellaneous	28	44	32	51	31	49	63
Total	242	35	312	45	384	55	696

Experimental studies have shown that old experimental animals had a very significantly higher mortality rate on exposure to stress than young ones. Furthermore, the adaptation to stress was less effective in the old than in the young ones. Finally, certain functional changes observed and measured in our experiments were mediated not by the endocrines but by autonomic nervous mechanisms. It was concluded that resistance to stress depends on an integrative mechanism that comprises adreno-medullary, adreno-cortical, pituitary, and hypothalamic functions and that this integration is interfered with by the process of aging (Grad & Kral, 1957). Clinico-anatomical observations, on the other hand, have demonstrated that an amnestic syndrome occurs in close connection either with lesions involving that part of the centrencephalic system that is now generally believed to be related to the level of consciousness, particularly when the mammillary bodies are involved, or with lesions, mostly bilateral, of the structures that compose the "harmonious mechanism which elaborates the functions of central emotions" of Papez (1937) and which today are subsumed under the name of the limbic system. The mammillary bodies that are linked via the fornix with the hippocampus form an essential part of Papez's harmonious mechanism. Both localizations have been found involved in cases with the senile amnestic syndrome (Kral, 1956).

It would appear possible, therefore, that the "malignant" type of senescent forgetfulness owes its ominous prognostic value to the fact that the underlying process, the senile brain disease, directly or indirectly interferes with the function of the centrencephalic system, where apparently also an important part of the integrative mechanism which enables the organism to withstand and master stress is localized.

The amnestic syndrome, however, is not the only type of memory impairment that can be found in senile dementia. When the disease progresses, the so-called "partial memories" of Meyrat (1944) also suffer. A patient may not remember, even after only 1 or 2 seconds, the fact that he had been shown an object, but he may still remember that he had heard a sound or had made a movement or vice versa. Eventually, all areas of sensory and kinesthetic function become involved, with one notable exception: the memory for pain sensation. These interesting findings could be made only in cases of senile dementia and not in other conditions characterized by an amnestic syndrome, such as alcoholic or traumatic Korsakoff, memory loss after ECT, or the postlobotomy amnestic syndrome. It was concluded that the loss of "partial memories" is essentially independent of the amnestic syndrome per se. Rather, it seems indicative of the extensive involvement of the cerebral cortex in addition to the limbic system (Kral & Durost, 1953). Although this hypothesis still requires confirmation in cases of forgetfulness with extensive cortical involvement of other etiology, such as general paresis, it is possible that the severe impairment of partial memories in senile dementia may be one of the factors responsible for the recent findings by Roth, Tomlinson and Blessed (1967) that in severely demented cases, diagnosed as senile dementia, the correlation between psychological and pathological findings (more specifically senile plaques) so sharply declines.

Summary

Two main types of memory disorders, both related to advancing age but different as to symptomatology and course, can be found among the aged. The "benign" type does not carry a bad prognosis. The "malignant" type was found significantly correlated to shortened survival time and increased death rate. All subjects with this type of forgetfulness developed a senile amnestic syndrome, necessitating institutional care. However, there was no significant difference in the causes of death between the two groups. It is concluded that the resistance to stress declines faster in patients with the senile amnestic syndrome than in aged patients with other forms of psychopathology or in well-preserved old people. In cases of senile dementia, severe impairment of partial memories also develops in addition to, and apparently independent of, the amnestic syndrome.

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A PSYCHOMETRIC STUDY OF VARIOUS MEMORY DEFICITS ASSOCIATED WITH CEREBRAL LESIONS

Jacques Barbizet and Elisabeth Cany

The subjects of the present study were patients with brain lesions of varied etiology causing diverse disturbances of cerebral function of which disorders of memory may or may not have been among the most prominent. We conducted a systematic investigation of their capacity to acquire and retain information in an effort to answer the following questions.

What is the ability of patients with different types of brain lesions to learn new verbal and new visual material?

Do such patients present consistent clinical profiles?

If so, can these profiles be related to the sites of the patients' lesions?

I. Method

We used a battery of memory tests with which we had acquired considerable experience in our Center of Reeducation in Memory and Language. The battery is designed to provide a systematic evaluation of a subject's intellectual functions: It measures his ability to increase his store of knowledge and analyzes his limitations in this area. In order to cover each of the principal aspects of intellectual function, we analyzed patients' memory capacities in relation to specific areas, for example, language or praxognosic (perceptual-motor) function. Furthermore, we spaced our tests in time, measuring the amount of material retained 1 hr, 24 hr, and 1 week after original learning and thereby were able to determine the temporal course of retention. Finally, we analyzed separately their capacities of immediate memory, immediate recall, learning, and retention.

Some of the tests in our battery are widely used, for example, the digit span and the word span; others were developed for our own purposes. All of the latter have been administered to normal subjects, but the samples are too small to allow us to establish norms for each age range and educational level. They are given merely to serve as points of reference and should not be taken as absolute criteria. Moreover, we have only measures of central tendency and no indices of variability.

A. TESTS

1. *Auditory Stimuli, Verbal Responses*

a. Digit Span A. The subject is required to repeat, in their original order, lists of digits of increasing length. The mean score for 105 medical students ranging in age from 18 to 24 was 6.40 digits with a standard deviation (SD) of 1.27; for 51 normal subjects over 65 years old, the mean score was 4.77.

b. Digit Span B. This test differs from the one just described in that each list consists of the items that made up the previous list, plus one additional digit: for example, 9, 2; 9, 2, 7; 9, 2, 7, 4; . . . . The score obtained on this test tends to be somewhat higher than the standard digit span, since learning effects carry over from one list to the next. A group of 88 medical students averaged 9.06 (SD = 2.12); 51 subjects over 65 years old obtained a mean score of 5.87.

c. Story. Several stories containing 22 units of content were each presented for immediate recall and for recall after delays of 1 hr, 24 hr, and 1 week. An example is the following:

A lion,/named Sultan,/escaped from his cage/through a door that was left unlocked/by a careless keeper./ A large crowd of spectators,/ visiting that Sunday,/escaped to the neighboring buildings./ A woman/dressed in blue/dropped her son,/one year old,/whom she had been carrying in her arms./The lion seized him./ The woman went back/and in tears/implored the lion to return her infant./The beast looked at her/with fixed eyes/for a long while./Finally, he let the child free/without having done him the least harm./

The subject's instructions were to listen carefully to the story he was to hear, and after it was over to repeat it as accurately as he could. His performance was scored for the number of units recalled and for the time taken to recall them. Medical students averaged 14.2 units on immediate recall; the mean score for a group of normal subjects 50-64 years old was 11.8, and it was 9.5 for a group of subjects who were over 64. (For delayed retention scores obtained by the second group, see Table I.)

Table I

Average Number of Content Units Recalled from a 22-Unit Story by Five Groups of Subjects at Various Times After Its Original Presentation

	Retention interval (hr)			
Group	0	1	24	168
Normal	11.8	11.0	10.8	10.8
Right hemisphere	13.0	11.4	10.4	10.6
Left hemisphere	5.8	5.2	4.6	4.4
Alcoholism	8.0	5.4	3.4	2.5
Dementia	5.7	2.3	1.5	0.7

d. Rey's Word List. A list of 15 familiar words is read aloud to the subject five times in succession, with a test of free recall after each presentation. The interval allowed for recall is 90 sec, and before each trial the subject is told how many words he recalled correctly on the previous test with the suggestion that this time he will do better. His performance is scored for the number of words correctly recalled, for intrusions, for words repeatedly recalled, and for the total time taken to recall the lists. This procedure is followed by a recognition test, in which the subject is presented with a story that contains all of the words that were in the list, his task being to identify them. Control norms are given by Rey (1958); the median score obtained by a sample of normal young adults was 65 items recalled over the course of five trials. For a comparable group of normal older subjects, the median score was 42.

2. *KIM Test (Visual Stimuli, Verbal Responses)*

The subject is presented with 20 familiar objects placed on a tray. He is given 90 sec in which to inspect and name all the objects, after which the tray is removed and he has to recall the objects. Next the board is shown to him again, with all those objects that he had correctly recalled on one side and the others grouped apart. Tests of delayed recall, without any further presentation of the material, follow after 1 hr, 24 hr, and 1 week. Performance is scored on each test according to the number of items recalled and the time taken to recall them. Control norms for number of items recalled are given in Table II.

Table II
Average Number of Items Recalled from the KIM Set by Five Groups of Subjects at Various Times After Initial Presentation

Group	Retention interval (hr)			
	0	1	24	168
Normal	13.2	14.6	14.0	13.0
Right hemisphere	10.2	11.2	10.3	9.2
Left hemisphere	8.0	6.9	6.4	5.6
Alcoholism	7.9	4.4	3.6	3.0
Dementia	5.5	4.6	3.0	2.4

3. *7/24 Test (Visual Stimuli, Motor Responses)*

The subject is presented with a checkerboard with 24 compartments, within which seven pieces are placed in a random pattern. His task, for which he is provided with a blank checkerboard and nine pieces, is to reproduce the original pattern after the first board has been removed. He is allowed up to 15 repeated trials, each with a display of 10 sec, to learn the pattern. Test of delayed reproduction (or of relearning if the first attempt is imperfect) follow after 5 min, 30 min, and 24 hr.

At each stage, performance is scored for (1) the number of pieces correctly placed on the subject's first attempt, which provides a measure of immediate memory or of the visual memory span, and (2) the number of presentations required for the subject to reach a criterion of perfect learning. If a subject fails to learn the pattern in 15 trials, after a week's delay he is given a simpler pattern made up of five pieces; and if he fails on that task, after a further delay he is presented with a pattern of three pieces on the same board. For subjects ranging in age from 41 to 79 years (with a mean age of 58), an average of four items is retained from the seven-piece pattern after one trial.

B. ANALYSIS OF DIFFERENT ASPECTS OF MEMORY

The acquisition of new information, whether it be verbal or visual or of another sort, reflects the operation of four psychological processes, which can be described as follows.

1. Presented only once with a limited amount of information, how much of it can a subject retain without error? The largest amount retained determines the limits of his *immediate memory* capacity.

2. Given a supraspan amount of information, how much of it does a subject retain after a single presentation? This measure varies both with the number of items presented and with the time taken to present them. The task involves processes of selection, structuring, and reorganization of the material. We denote performance on these complex tasks as measuring *immediate recall.*

3. How can a subject increase the amount that he retains over repeated study trials? We may distinguish between two phases of learning: (a) *partial learning,* after a limited number of repetitions, in which level of performance measures degree of learning, and (b) *complete learning,* in which the performance is constant and only the number of trials can vary.

4. How long does information persist in memory? By means of successive tests of the material previously presented, we determine a forgetting curve, or a measure of *retention.*

In order to measure these four processes, the following tests were selected, the different stages of each test reflecting different stages of the mnestic process:

Immediate memory: Digit Span A; 7/24 Test
Immediate recall: KIM Test; Story
Learning: Digit Span B; Rey's Word List; 7/24 Test
Retention: Story; KIM Test; 7/24 Test

C. SUBJECTS

These tests were given by a team of psychologists to 120 patients hospitalized for cerebral lesions. They included 30 patients with left hemispheric lesions, with or without aphasia; 30 patients with right hemispheric lesions; 42 patients with neurological disease of alcoholic origin; and 18 patients with presenile or senile dementia. Their age distribution is given in Table III. In addition to our battery, these patients also were subjected to systematic tests of language and praxognosic function.

Table III
Number of Patients Per Age Group

Diagnosis	20-30	31-40	41-50	51-60	61-70	71-80	Total
Left hemispheric lesion	–	6	3	5	6	10	30
Right hemispheric lesion	1	–	4	15	8	2	30
Alcoholism	1	7	17	13	4	–	42
Presenile and senile dementia	–	3	1	1	7	6	18

II. Results

We shall present our results separately for each group of patients.

A. LEFT HEMISPHERIC LESIONS

Our sample was made up of 30 patients with left retro-Rolandic hemispheric lesions. They included 22 spontaneous vascular lesions, two traumatic intracerebral hematomas, and six tumors. The patients were examined within 3 months to 1 year from the onset of their illness. The unilaterality of their lesions was determined by neurologic examination. Of the 30 patients, all right-handed, four showed no clinical signs of aphasia; the remaining 26 patients had aphasia that was quite manifest, but not so severe as to prohibit them from performing on our memory tests. Their performance scores on the several tests are shown in Tables IV and V.

From the analysis of the data a defect in the memorization of verbal material is obvious, as is shown by: (1) a diminution of the digit span,

Table IV
Average Scores Obtained on Various Memory Tests by Patient with Left Hemispheric Lesions

	Degree of aphasia				
Test[a]	0	1	2	3	Average
Span A (4.77)	5.1	4.4	3.9	2.9	4.41
Span B (5.87)	6.3	5.1	4.5	2.9	4.70
Story (11.4)	8.8	7.2	4.2	2.0	5.70
Rey's List (39-56)	32.0	28.9	14.0	3.8	19.68
KIM (13.2)	10.4	6.8	5.5	6.3	8.03

[a] Figures in parentheses represent control norms.

Table V
Average Number of Elements Recalled by Three Groups of Subjects on Test 7/24 (Seven-Element Pattern) after Various Delays Following Initial Mastery of the Task

	Retention interval		
Group	5 min	30 min	24 hr
Normal	6.16	6.26	6.06
Left hemisphere	5.23	5.16	5.02
Right hemisphere	5.90	5.66	5.60

the more significant the more severe the aphasia; (2) a diminution in immediate recall; and (3) a defect in verbal learning. In contrast, verbal material that has been apprehended is well maintained in memory and correctly recalled 24 hr or a week later.

The defect in memorization also affects visual material. While 100% of the normal control group was able to achieve perfect performance on the 7/24 Test, only 61% of the left hemispheric patients were able to do so. Of these patients, 25% were able to master the task when the number of items was reduced to five and 9% when it was reduced to three. The number of items correctly recalled on the first trial with the seven-element board was 3.15 for the patients and 4.00 for a normal control group. The recall scores after initial mastery of this task were 6.16, 6.26, and 6.06 for normal controls after intervals of 5 min, 30 min, and 24 hr. For the patients, the comparable scores were 5.23, 5.16, and 5.02. Retention functions for the story and the KIM tests are shown in Tables I and II for all four patient groups and control subjects.

Comment: A deficit in the memorizing of visually presented information is evident in patients with left hemispheric lesions. It shows up both in a decline of immediate visual memory and in difficulties in learning. Retention is only slightly inferior to that of normal control subjects, and the difference is relatively stable, representing only a very moderate loss in patients who were able to master the seven-element pattern on Test 7/24. In contrast, among the patients unable to master this task, the retention was also very poor.

Whether for verbal or visual material, the memory capacity of patients with left hemispheric lesions appears to lie far below the control norms, both in regard to general level of achievement and time required for learning. Performance on all tests is affected, whatever function they measure. There is a twofold deficit in memory, verbal and visual. This deficit primarily affects the processes of putting information into storage; if those are successful, retention is satisfactory and there is practically no forgetting.

B. RIGHT HEMISPHERIC LESIONS

Of the 30 patients studied, 26 had vascular lesions, two had traumatic hematomas, and two had tumors. These patients were selected with a view to the unilaterality of their lesions as established by neurological examination. Most of these patients presented an apraxognosia of varying severity. On the basis of the Kohs test described by Goldstein and Scheerer (1941), they were ranked into four groups in order that we might obtain a better estimate of their base levels. The results were evaluated according to qualitative and quantitative criteria (type of error and number of successes, respectively). In contrast to patients with left hemispheric lesions, those with right retro-Rolandic lesions showed constant deficits *only* in the visual tests of our memory battery, as shown in Tables V, VI, and VII.

Inspection of these tables suggests the following conclusions: (1) Patients with nonapraxic right-sided lesions differ very little from the normal population, except for their low percentage of success on Test 7/24. Averaging across all these patients, 44% were able to achieve perfect performance on the seven-element task (as compared with 100% of the subjects in the control group). When the number of items was reduced to five, 20% were able to master the task; and when it was reduced to three, 26% were able to do so. (2) An analysis of successes on the several boards, in spite of some scatter, shows that the patient's degree of apraxia exerts a strong influence on his learning of this task. This conclusion is further confirmed by an examination of the time taken for learning. (3) As with left hemispheric patients, retention is somewhat inferior to that of the control group. Right hemispheric patients who do not fail in initial learning forget very little, and their forgetting curve is only slightly steeper than that of the control group.

In contrast to the difficulty they have with visual material, patients with right hemispheric lesions remember verbal material quite normally, as indicated by the data shown in Table VII. It seems paradoxical that their immediate recall of the story was superior to that of the normal control group matched for age and educational level. Without proposing a definitive explanation, we suggest that this result can be attributed to a verbal compensation for visual deficit. Indeed we noted that these patients display very high motivation and sustained attention when called upon to exercise functions they have preserved.

Table VI
Number of Patients with Right Hemispheric Lesions Mastering Test 7/24 as a Function of Complexity of the Task and Degree of Apraxia

	Degree of apraxia				
Number of elements	0	1	2	3	Total
7	5	6	2	–	13
5	1	2	3	–	6
3	–	1	4	3	8
<3	–	–	–	3	3

Table VII
Average Scores Obtained on Six Psychometric Tests by Each of Six Subject Populations

	Test					
Group	Span A	Span B	Story	Rey's List	KIM	7/24
Normal	4.77	5.87	11.40	39–56[a]	13.20	4.00
Left hemisphere	4.41	4.70	5.70	19.68	8.03	3.15
Right hemisphere	5.20	*b*	13.00	47	10.20	2.83
Alcoholism	4.90	6.09	7.76	32.92	9.10	3.16
Polyneuritic	5.16	6.47	10.14	39.42	10.19	3.66
Encephalopathic	4.64	5.71	5.38	26.42	8.04	2.66
Dementia	4.33	4.69	5.80	25.00	5.60	*b*

[a] The upper and lower values are for younger and older subjects, respectively.
[b] Not available.

In conclusion, the amnesia for visual material observed in patients with right hemispheric lesions seem to be closely related to their apraxia. It is manifested in defective immediate memory for visual material and in difficulty in learning such material. Once learned, however, the material is rather well retained, except when a massive apraxia completely disrupts the process of recall.

A comparison of the test results for patients with left and right hemispheric lesions clearly demonstrates that the memory deficit is always secondary to the functional derangement. In those with left-sided lesions, who are impaired in language function and only to much lesser extent in praxognosic skills, the memory deficit for visual material is relatively very small. Patients with right-sided lesions who suffer from defects in praxognosic skills show deficits of learning only in that area.

These instrumental amnesias, which are associated with focal lesions, primarily affect the processes of apprehension and acquisition. To the extent that information is stored, it also will be retained. It should be noted that this is so only if the instrumental deficit is not total; when it is, the absence or severe impairment of apprehension does not enable one to measure retention. This conclusion is clearly illustrated by those left hemisphere patients who were affected by a massive aphasia and whom we had to exclude from our sample because they were incapable of comprehending the verbal symbols included in our tests. We did not have to reject any of the right-hemisphere patients, and therefore their rate of failure on the seven-element pattern of Test 7/24 (56%) seems more significant than the corresponding rate in patients with left-sided lesions (39%). It is likely that our third patient group, which showed severe apraxia on the Kohs test and failed on the five- and seven-element patterns of Test 7/24, had important lesions in the right parieto-temporo-occipital area, comparable in extent to those in the left hemispheric patients who had to be excluded from our study.

It is worth noting that the various tests of immediate memory used since Jacobs (1887) are selectively sensitive to certain localized hemispheric lesions. For example, immediate memory as measured by the number of digits, syllables, or words reproduced after a single presentation is specifically altered by lesions affecting the *instrumental language* area in the dominant hemisphere. At the same time, immediate visual memory—as measured by the number of visual elements retained after a single trial of a given duration—is affected by all lesions in the right or left hemisphere that are accompanied by an aproxoagnosia.

Consequently these tests, rather than measuring any general disorder of memory, instead may serve to reveal specific localized disorders, and they tell us not about memory in general but about the cerebral functions that must be intact in order for structured information to be immediately apprehended. By means of these tests of immediate memory, we in fact measure a subject's capacity to retain several items simultaneously. Such tests thereby allow us to study the psychological functions that depend on certain cortical association areas.

The memory tests presented to our brain-damaged subjects allow us to measure various instrumental defects quite precisely even when these defects appear clinically to be very diverse. Instead of relying on qualitative evaluations, we have obtained global measures of learning capacities and performances in certain specific areas. Moreover, our method of studying memory can reveal not only an instrumental deficit but may considerably augment its signs, emphasizing them in a manner that brings to the surface latent defects that are not clinically ascertainable. It enables us, moreover, to determine the extent to which a function has deteriorated. The present study in this way can contribute to research on latent verbal and apraxic defects.

C. ALCOHOL ADDICTS WITH NEUROLOGICAL LESIONS

In half of our group of 42 alcoholics, a sensorimotor polyneuritis constituted the major clinical sign. In the other 21 cases, mental disturbances persisting after an acute confusional episode resulted in a diagnosis of alcoholic encephalopathy. The distinction between polyneuritis and encephalopathy seems arbitrary in view of the fact that both peripheral and central neurological signs, and as well as mental disturbances, may be present simultaneously in the same patient. In fact, if one classifies these patients according to their performance on various psychometric tests, one finds a continuous gradation from higher mental processes that are virtually normal at one extreme to profound alcoholic dementias at the other.

Our sample was composed of 29 women and 13 men. Their mean age was 48. The two subgroups did not differ with respect to average cultural level. All the patients were tested in the chronic phase of their amnesic syndrome (Talland, 1965), 4-18 months following the initial confusional state. Their scores on the various tests administered to them are shown in Table VII, separately for those patients with peripheral signs (the polyneuritic subgroup) and for those with central disorders (the encephalopathic subgroup). The retention scores shown in Tables I and II, however, are based on both subgroups' performances.

Comments: Patients classified on clinical evidence as encephalopathic displayed poorer performances on all tests than those classified as polyneuritic. The latters' immediate memory capacity is normal, their immediate recall is slightly impaired, and their ability to learn as measured by Rey's List and the 7/24 Test is quite mediocre (see Table VII). Insofar as they show a deficit, it applies to verbal and visual material alike. Delayed retention tested after 1 hr, 24 hr, and 1 week, both with the story and the KIM test, shows some forgetting. It amounts to 33% at the longest interval.

There are considerable individual differences within this group: Some patients, whose immediate recall is good, retain the material almost normally, while others forget very rapidly. Although not very numerous in our total sample, the latter patients are of particular interest in that they demonstrate a special type of amnesia, a dissociation between immediate memory and delayed recall along with a rapid rate of forgetting. As an example, let us cite the case of a 50-year-old secretary who showed no significant deterioration of normal intelligence as measured by the Wechsler–Bellevue Scale and showed normal immediate memory (a score of 6 on Digit Span A) and normal immediate recall (scoring 11 on the KIM Test and 14 on the story) but who in the tests of learning fell far below average, scoring 32 on Rey's Word List as against a norm of 56. Her retention after 1 week amounted to 3 units on the KIM Test and to zero on the story.

This type of deficit, marked by good immediate retention of visual or verbal material but of very rapid forgetting, is known to be associated with a bilateral lesion of Papez's circuit which, in alcoholics, occurs typically at the level of the mammillary bodies. Indeed, this type of amnesia, which is equally characteristic of bilateral hippocampal resection—hence our proposal to define it as hippocampomammillary amnesia (Barbizet, 1964, 1966)—rarely occurs in a pure form.

In patients with encephalopathies of alcoholic origin there is often a wide gap between clinical observations and the more exact data derived from psychometric testing. A number of our patients behaved almost normally, could dress themselves, found their way to the cafeteria, debated with their fellow patients, and occasionally played a game of cards or bowled in the garden. Their memory disorder was quite apparent; not only was their recall of many past events of their lives deficient (especially of events that occurred during the years immediately preceding their illness), but they also were incapable of acquiring new information, for example, the name of a person in their daily surroundings or the names of their visitors or notable political events they had an opportunity to learn about from newspapers or television. If shown some object, they described it quite correctly; but if it then was hidden from them, in a few minutes they neither remembered the object nor its being hidden from them.

Apparently in these patients the memory disturbance is manifested in a pure and possibly isolated form; their familiar skills, verbal abilities, and judgment were within normal limits. This latter impression, however, was often contradicted by psychometric tests that showed evidence of deterioration in the use of learned information. In familiar situations these patients responded with stereotyped verbal or other behavior and proved incapable of combining the several stages necessary for the completion of a complex task or of learning to perform such tasks even after repeated attempts.

In our group of 21 patients with an encephalopathy of alcoholic origin, immediate verbal memory was relatively well preserved, but immediate visual memory was considerably impaired, as was immediate verbal recall. They also were unable to organize effectively the material presented for learning. The retention functions for the story and KIM Test based on all these patients indicate that the amount forgotten is indeed considerable. But note that the initial points of the functions lie well below those for the normal control group. This result seems to indicate diffuse brain lesions and suggests that in addition to the damage in the mammillary bodies so often found in these patients there is also general cortical damage. Hence we propose the name *global* or *mixed amnesia* for this type of amnesia.

To conclude, of our 42 alcoholic patients affected by a neurological disease, some are remarkable for their inability to retain that which they initially apprehend, and in most cases this defect in retention is also associated with a deficit in the ability to store information in memory.

D. Presenile and Senile Dementia

There could be no doubt regarding the diagnosis of dementia in this group of 18 patients. From an etiological point of view the group was not homogeneous, since it included two cases of infectious encephalitis, three of vascular dementia, and 13 of senile dementia. Nor was it homogeneous in regard to symptoms: The patients' only common denominator was in fact their dementia. They were all too severely deteriorated to be able to live on their own but not so severely as to prevent their being tested—although the former fact created difficulties at times and made it difficult to interpret their performance scores. On the digit span their mean score was 4.33 as against 4.77 for the normal control group; on the KIM Test they scored 5.6 and on the story 5.8, as compared with 13.2 and 11.4, respectively, for the control group. Their scores were 4.69 on Digit Span B and 25 on Rey's Word List as against 5.90 and 39, respectively, for the control group. Their delayed recall scores are shown in Tables I and II.

Comments: A deficit in Span A becomes very significant for immediate recall, learning, and retention. In these patients no performance shows intact function; memory function seems to be impaired in its entirety and at every level of structuring. It is of interest to compare the forgetting functions for the alcoholic and the demented group on the KIM Test and the story. If the latter group seems to be more severely affected, it is also evident that the two functions indicate different degrees of dementia, and more specifically in the alcoholic group (which also was younger). This result also corresponds with the anatomical data, the neural damage being far more extensive in patients affected by massive and global amnesia.

We should have liked to complete this study with a report of results obtained on memory tests given to a large group of patients with frontal lobe lesions. Although we have studied some 25 with frontal lobe lesions, our findings are not sufficiently homogeneous to constitute a reliable set of data. Whether the patient suffers from a frontal meningioma, a frontal glioma, spontaneous or traumatic hematomas, frontal lobectomy, or Pick's dementia, in most instances it is impossible to determine the exact anatomical damage. On the other hand, the variety of clinical syndromes presents too diverse a picture to enable comparisons to be drawn.

Nonetheless, a survey of our records allows us to draw certain conclusions that are presented quite tentatively here. Patients with frontal lesions always show poor learning, and the disability is greater the more extensive is the lesion. This deficit applies to verbal and visual material alike, and we have found no difference in that respect between patients with left or right frontal lobe damage. The source of the defect is in the ineffective apprehension of the information presented for retention: The patient is unable to explore the situation efficiently in order to place the material in memory, he rapidly grasps some items which he keeps on repeating accurately enough but in no systematic order, and he quickly loses these fragmentary and poorly structured memories. In patients who are only mildly affected, given that they have succeeded in mastering Test 7/24, a geometric figure, or a simple story, long-term retention can be excellent. The poor learning capacity of patients with frontal lobe lesions seems to us to stem essentially from their difficulties in apprehension, in organizing and recalling the information presented to them, which are more pronounced the more complex is the task. One encounters, at a different level of function, the same specific amnesias as are associated with right and left hemispheric lesions.

III. Conclusions

Our study of the effect of different types of brain lesions on the acquisition and retention of verbal and visual material allows us to draw a number of conclusions:

1. Instances of apparently isolated defects in retention, in patients who otherwise exhibit no mental deterioration, are rare. They have been observed in some alcoholics diagnosed as having Korsakoff's

disease; such patients are apt to forget completely within a few minutes or a few days information that they initially recalled correctly.

2. Far more frequent is a defective ability to put information into storage, as manifested in poor immediate memory, immediate recall, and learning. Lesions localized in the retro-Rolandic area of either hemisphere cause such deficits in registration and in learning.

In patients with left hemispheric lesions, whose language functions are disturbed and whose praxognosic skills are affected to a lesser degree, memory for verbal information is defective and memory for visual information is affected only to a relatively small extent. Patients with right hemispheric lesions, whose praxognosic functions are impaired, show memory defects in that area only. In these instrumental amnesias, information that is initially well grasped is also well retained. Of course, this is true only of instrumental amnesias that are not total, because in the absence of original comprehension forgetting cannot be meaningfully tested.

3. Global amnesias associated with diffuse cerebral damage—for instance, those which follow alcoholic or infectious encephalopathies or occur in presenile and senile dementias—are characterized by severe difficulties in registering verbal and visual information and by poor retention or, indeed, no retention at all. In these cases psychometric testing allows for a quantitative evaluation of a patient's residual memory function, the extent of the deficit and, via repeated tests, its stability over time.

The rapid forgetting of information that was correctly perceived indicates a bilateral change in the long tract of axonal connections that is known as Papez's circuit. The latter's integrity seems to be necessary both for the integration of new information with old, which is the process of initial learning, and for the repeated application of old memories that is required for the effective execution of sequential task or for the ability to conduct a coherent conversation. In contrast to lesions along this interhemispheric circuit, lesions that are localized in one hemisphere destroy certain engrams and disturb communication between those that are intact, altering verbal or praxognosic functions and impairing learning of one sort or another.

The integrity of the important functional system that has progressively developed in everyone's brain over the course of repeated learning since childhood is necessary for the exercise of these complex functions known as memory, which enables everyone to learn and to utilize a large array of information and which are indispensable for its retention.

There remains much that is unknown, and a systematic investigation of changes in different aspects of memory by means of reliable psychometric tests can best provide the data to be checked against neuropathological and biochemical observations.

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MEMORY IN TEMPORAL LOBE EPILEPTICS[1]

Dietrich Blumer and A. Earl Walker

Memory disorders associated with seizures are difficult to study precisely and systematically. Perhaps for this reason the literature on the topic is extremely sparse. However, it is known that the impairment of memory is different in the various forms of epilepsy. Almost invariably there is no memory reported of a *grand mal seizure*. Consciousness may be lost before the seizure begins its course or a memory trace may not be established. This type of attack is usually followed by a progressively clearing retrograde amnesia, as well as by a postictal amnesia of variable degree. If generalized attacks take place in succession, the period of memory impairment may increase very markedly. However, we have little interest in the amnesia of grand mal attacks, because it gives us no information about the role of discrete parts of the brain in memory.

In simple *focal motor cortical seizures*, the lack of change in consciousness and memory is remarkable. Typically, a patient who has his first focal motor seizure vividly remembers how he tried to pull down the rising arm with the unaffected limb. We have, in addition, one patient with focal motor attacks who reported full awareness of events throughout the entire generalized phase of the seizure, which lasted 2–3 min.

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In the case of *petit mal lapses*, careful studies have documented a brief retrograde amnesia of a few seconds duration (Jus and Jus, 1962). Characteristic of petit mal, however, is the ease with which the thread of a conversation can be promptly resumed following the attack.

Patients who experience *minor seizures due to a principal epileptogenic focus in either the prefrontal or temporal lobe areas* appear to be similar in their memory impairment. Because we have studied only a small number of patients with frontal seizures, we will exclude them from this presentation. Instead we will discuss our findings about memory disorders in *temporal lobe epilepsy*. Mnestic disturbances and changes of consciousness are the outstanding traits in this form of partial epilepsy.

We reviewed the histories of seizures and our own observations of seizures in 60 temporal lobe epileptics who had been admitted to the neurosurgical division of the Johns Hopkins Hospital. Our efforts to analyze the type and extent of memory impairment in this material met with a number of difficulties. Frequently it was not clear what the patient recalled of his own seizures, what he had learned from others about his attacks, and what others reported as their observations. Even the astute observer cannot exactly measure the degree to which consciousness may be impaired, a measure important for our understanding of the memory impairments. Retrograde and postictal amnesia can only be well estimated by an active on-the-spot investigator. Despite this, the presence of memory impairment associated with the seizure could be clearly established in all but five of the 60 patients.

Let us first of all review which events of the seizure are remembered. In our sample of 60 patients, seven frequently were completely unaware that a seizure had taken place. Five of these patients had complex automatisms, and all but one almost never experienced an "aura." The aura indeed stands out as a warning for events to come and as a signal that events have taken place. The aura is the remembered initial part of the seizure, or the *primictal phase*. The *ictal phase* as used in this paper refers to the part of the seizure not consciously experienced or not recalled.

Forty-five patients reported the experience of an aura that could be variously classified as autonomic (epigastric), special sensory (including vestibular), psychic, or somatosensory in type or as impairment of consciousness, in this order of frequency. The one trait that may be common to all these experiences is their undefinable nature: a "peculiar feeling in the stomach," a "funny feeling all over," an "undescribable taste or odor," a feeling of unreality or strangeness, or a feeling that something dreadful was going to happen. The vagueness of events remembered is further apparent if a patient is unable to recall with certainty the correct sequence of sensations during a complex primictal phase.

One of our patients would suddenly feel dizzy and at the same time hear a buzzing in her ears that would turn into her mother's voice, repeating monotonously "You are going to have a sicky feeling." This she remembered clearly, and these events can be considered part of the primictal phase. However, complex phenomena such as auditory hallucinations are more often not well remembered and may be considered part of the ictal phase proper of the attack. Thus, an adolescent girl regularly heard quarreling voices during the attack but could not recall the conversation. Similarly, another patient reported that the same thoughts seemed to occur to him with each attack, but he could not recall them. Another patient heard a familiar song, but could not place it. A fourth patient would hear, as the sole manifestation of the attack, a rhythmic tune, sometimes 20 or 30 times during one day. She could hum the tune while she heard it, but could never reproduce it an instant later. A tape recording was made during an attack; on replay she recognized her humming of the tune as something "a little familiar" to her. In another case, a young woman had seizures consisting of a series of peculiar sensations. During her attacks she would write notes to herself stating how she felt. For example: "Everything seems strange. Perspiring. Feel stuffy. Everything seems so casual, and I am more or less disinterested." Later she would be unable to recall what she had done even when she read it on paper.

Common observable signs of the seizure, such as staring, facial movements, manual fumbling, other motor manifestations, and loss of postural reflexes are usually not recalled and constitute part of the ictal phase. A majority of our patients (34) displayed typical automatisms consisting of more complex motor activity with or without speech, or speech alone during their attacks. We found only five patients who occasionally—and never regularly—reported fragmentary and vague memories of these automatisms. One patient stated that he remembered events of a certain seizure "like a dream." Another patient, who would mumble during the attack, recalled with surprise on a single occasion that she had heard herself mumble without knowing what she said.

In the following case the automatism was surprisingly well remembered.

Case 1. A 20-year-old computer operator experienced his customary aura consisting of "chills or funny feelings" over his whole body.

He then dropped a stack of IBM cards, knew that they fell on the floor, but for some reason knew he could not pick them up. He walked to another part of the room, knew he was walking, but not why. He stood there for a few seconds, then "snapped out of it." He asked bystanders about his actions and they confirmed what he had experienced himself.

Behavior during an automatism typically is inappropriate, aimless, fragmented, and often repetitive. Seemingly purposeful actions and occasional responses to the environment are quickly aborted. Speech during the automatism is more often observed in patients with right temporal foci. Like the behavior, the speech is inappropriate or nonsensical, incoherent, and frequently consists of repetitive utterances. Typically, the automatism is of brief duration. Some examples of so-called automatisms in which the behavior was perfectly appropriate but not remembered are discussed later.

We then can roughly separate the more simple events of the primictal phase, which are remembered, from the more complex and at best vaguely remembered events of the ictal phase. This suggests that the memory process fails with spread of the seizure discharge. It is, of course, a common occurrence that the same patient who usually can recall the events of his *brief* seizure will be amnesic should he experience an automatism. On the other hand, brief amnesia may occasionally be the only signal of a seizure. A memory lapse, a forgetting of what was just talked about, appeared to represent a seizure in three patients.

Following a brief seizure, patients with temporal lobe epilepsy may be able to take up a conversation or activity promptly and precisely where they left it. At least this may be attested to by lay observers. More commonly, a period of what is referred to as confusion follows upon the attack; this seems to be the rule following more severe attacks. The postictal confusion appears to be due to (1) a still narrowed consciousness, (2) a retrograde amnesia, and (3) a postictal amnesia.

The *retrograde amnesia* could be well documented in the following instance.

Case 2. A 24-year-old male suffered a seizure of about 1-min duration in the presence of the interviewer. He showed a loss of postural reflexes and mild trembling of his limbs, followed by some fumbling with his pajamas. He then walked about in a daze, but gradually began to respond in a more appropriate manner. When the phone at his bedside rang, and the interviewer told him his father wanted to say hello, he acknowledged this. By now, less than 5 min since the attack, he knew who the interviewer was and why he was with him. But he was unable to recall a single topic of the 15-min conversation that had preceded the attack, although he was asked repeatedly about it. When asked if he had shown something to the interviewer, he recalled that he had produced a newspaper clipping about a certain medication. As he went to his drawer he recalled a few other items he had shown, and this pleased him. He still did not recall anything else, but when he was given simple clues, he could develop the other topics of the conversation, such as seizures and schooling, up to the moment of the seizure, although some vagueness remained. He was asked if his girlfriends had been discussed with him—which was not the case— and he stated that he did not know. He recalled spontaneously only one topic, the very first one that had been covered: being tested by two different assistants. He did not remember that his father had phoned. This, then, is an example of postictal amnesia since the examiner knew that the event was perceived at the time.

In another patient the retrograde amnesia did not always clear up so promptly.

Case 3. A patient with ictal automatisms of 30–60 sec in duration reported that it would take him sometimes a few minutes, sometimes 2 hr, and at least on one occasion a whole day until his memory of events preceding the attacks returned. In spite of the marked retrograde amnesia, he was quite definite that he could always recall the funny feeling in the throat that represented his aura. Recently, since taking a new anti-convulsant pill, he reported that he could remember all events preceding an attack quite promptly.

One patient spontaneously reported an orderly shrinkage of the retrograde amnesia. He would remember more distant events first after a seizure, and the last thing he had done before the seizure would come back last.

In another patient the extent of the retrograde amnesia varied with the severity of the seizures.

Case 4. The patient would experience a funny feeling in the stomach going up to the throat and then his face would flush and he would frequently develop a "coppery" taste in the mouth. He remained fully aware during all his seizures. Following a left temporal lobectomy for astrocytoma, his attacks were milder—"not hitting the face so hard." His wife reported that he usually would ask what they had been talking about just before the attack, but that this slight forgetfulness also had become milder since the operation.

While some patients may be quite definite that they always remember the aura, in spite of the presence of a significant retrograde amnesia, we learned from one patient that he could not recall the aura when the seizure had been more severe than usual.

A diagnostic problem arises when episodes of perfectly appropriate behavior are reported but not remembered by the patients. Such behavior differs drastically from the poorly organized behavior of the usual automatism. Two of our patients reported such episodes. One of them had been able to drive a car with a passenger for about eight blocks, observing stop signs properly; but then he stopped, slumped over the wheel, and turned off the ignition. He had no memory for these events. On other occasions he had been able "to play cards automatically with his friends without anyone realizing that he was amnesic for the event." The other patient, on one occasion, had no recollection of doing his school work as usual for a period of perhaps 3 hr.

Neither of these patients had ever experienced the usual type of automatism. Both usually had brief attacks of "dimming-out" or "loss of concentration" followed by an inability to recall what had happened immediately preceding the attack. We therefore suspect that the episodes described are actually "pseudo-automatisms." They probably represent normal preictal behavior that was involved in an unusually extensive retrograde amnesia. The attack that caused the amnesia may be overlooked in such patients.

Postictal amnesia is even more difficult to document than retrograde amnesia. In the postictal phase the patient's responses are often poor and observers tend to avoid questions. The following is an observation of a patient with speech automatisms who continued to talk through the postictal phase.

Case 5. A 24-year-old male, about to leave the hospital, was explaining to the interviewer that he was going home by train, when he became unresponsive. At first he remained erect, but then he sat down and started to say "uh—uh" over and over again followed by, "I am not feeling well," and he pursed his lips as if he were about to spit. This was his customary seizure pattern, which he knew only from descriptions of others. After perhaps 2 min he began to respond. He said he had had a seizure and complained about a headache. He then expressed concern about the amount of anticonvulsant drug he was taking. He discussed his means of transportation to the train station when this topic was brought up by the interviewer. His talk was appropriate and coherent, except for one peculiarity: He would lose the thread of his conversation completely upon any distraction. At the end, the interviewer inquired what the patient remembered about the preceding 10-min conversation. It became clear that he had a total amnesia for the ictal and postictal events.

In contrast to retrograde amnesia, there seems to be no shrinkage of postictal amnesia. The question may be asked whether postictal amnesia may be more severe in patients with bitemporal foci and marked bilateral epileptic discharges, as was the case in the last-cited patient. It is evident that even a very mild attack, that is, the aura alone, may be followed by some postictal amnesia; patients often do not know if their seizure took its full course after the aura. In some circumstances this uncertainty may cause considerable anxiety.

The memory disorder in head injuries and in generalized convulsions—with retrograde and anterograde amnesia—is surprisingly similar to the memory impairment in temporal lobe seizures. This seems to point to the unique role of the temporal structures. Of course, our clinical study allows no conclusion regarding the importance for memory of structures outside the temporal lobes, which may become involved with the spread of the epileptic discharges. Psychological studies in patients with depth electrodes can provide a more definite answer. But there is still one phenomenon, unique to temporal lobe function, which has to be mentioned.

The *déjà vu*, an illusion of memory, is a well-known ictal phenomenon. It was reported by five of our patients, of whom four had a right temporal lobe focus. A person, an animal, an object, or an entire situation never seen or experienced before may suddenly appear familiar. One of these patients described that everything would seem "very strange but vaguely familiar." Another patient at times also experienced sudden feelings of unfamiliarity. A third patient stated right after a seizure, "It was like a dream of what I had seen before." A fourth patient at times "barely sensed the *déjà vu* phenomenon and the accompanying feel of fear." It is obvious that the *déjà vu* does not occur in a setting of clear consciousness. The twilight character of the *déjà vu* is in accordance with the nature of most of the memories of ictal events. This "hypermnestic" phenomenon seems to be strictly a primictal event, just as the amnesia is part of the following ictal and postictal phases.

Brief mention has to be made of the *interictal memory problems* of patients with temporal lobe epilepsy. Relatives may state that the patient has a poor memory but may indeed refer to ictal memory deficits or specifically to a period of time with very frequent seizures and consequent memory impairment. A number of patients who complain of a poor memory may be able to carry on their daily activities without difficulties, and on psychological testing they may show no significant memory impairment. If ictal episodes cannot explain the difficulties, one may be tempted to implicate "subictal discharges." This area still needs much further investigation.

A brief review of the findings in patients with *unilateral temporal lobectomy* adds considerably to the understanding of our topic. In our total of 60 patients, 42 underwent this operation. The 26 who continued to have seizures invariably continued to experience the same aura. This of course suggests that, at least in these cases, the aura does not arise in the anterior temporal lobe that was resected. Therefore, adjacent areas, the contralateral temporal lobe, or subcortical structures may be implicated in the production of the aura.

The operation may reduce either the frequency or the severity of the attacks. Three patients were freed of grand mal attacks but continued to have automatisms; five patients lost their automatisms but retained the aura or primictal phase. This reduction in the severity of the seizure patterns means that the period of amnesia is likewise shortened or eliminated. These findings indicate that unilateral temporal lobectomy can reduce the spread of the epileptic discharge and, in effect, reduce the amnestic period of the seizure.

Certain memory impairments may become apparent only after temporal lobectomy. These impairments are chronic and occur with full consciousness. On selected tests we could detect neither perceptual visual impairment in patients with right temporal lobectomy nor impairment of verbal comprehension in patients with left temporal lobectomy. The visual tests employed were the McGill Picture Anomalies and the Mooney Faces; the verbal tests were the Gorham's Proverbs and the Wide Range Vocabulary. But the verbal memory impairment of patients with temporal lobectomy on the dominant side, so well described by Milner (1958, 1962), could be confirmed. On the two verbal subtests of the Wechsler Memory Scale, the patients with left temporal lobectomy scored lower than those with the right-sided operation. In the Logical Memory subtest this difference became significant when recall was requested again after a delay of 90 min; in the Associate Learning Task there was a significant lowering in the number of words recalled by patients with left temporal lobectomy, with and without delay. A more complex verbal learning task developed by Weingartner (1968) regularly differentiated the left from the right temporal lobectomized patients. To evaluate the sensitivity of the test we had one of our new co-workers, Mrs. Nachbahr, make a blind lateralizing diagnosis on the basis of three sets of scores from the test. In 21 of the 27 cases examined, she correctly diagnosed the side of the lobectomy. In one case no diagnosis could be made, and in five the laterality was incorrectly judged. One patient, clearly "misdiagnosed" as having a "nonleft" temporal lobectomy, had had only a small resection of the left lateral temporal cortex and in contrast to all the other patients had not had an excision of the hippocampal area. Among the remaining four "misdiagnosed" patients, three were left-handed—the only left-handed patients in the entire group. However, we have no other evidence than handedness for suggesting reversed dominance in these cases.

Like lesser memory difficulties, global memory loss after unilateral temporal lobectomy appears more frequently in patients with left temporal lobectomy. Among the 42 patients who had been operated on, two patients with left temporal lobectomy suffered from a persistent significant memory impairment. A third patient in whom a left temporal astrocytoma had been only partially resected showed a severe global memory loss—comparable to the state after bilateral temporal lobectomies. Such patients possess a good immediate recall—their digit span may be excellent—but their memories do not consolidate and may disappear with the slightest distraction and events remembered are poorly localized in time. These memory impairments are indeed similar, if not identical, to those we have found in association with temporal lobe seizures. This is not a surprising result since the ictal and postictal paralysis of mesial temporal structures is comparable to a temporary absence of the same structures.

Finally, it may have occurred to the reader that we have come across something familiar to everyone. The memory in temporal lobe seizures—in so-called twilight or dreamy states—as well as the memory after bilateral lesions of the mesial temporal lobes is as vague, fragmentary, fleeting, or nonexistent as is the memory of our own dreams.

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TRAUMATIC RETROGRADE AMNESIA AND NORMAL FORGETTING

Moyra Williams

The existence of retrograde amnesia (RA) following organic illness and cerebral trauma has long been a stumbling block to theories of memory. Retrograde amnesia seems to defy all the laws of normal forgetting, so that to explain it, it has been found necessary to introduce a special concept—that of consolidation. The differences between RA and normal forgetting have in the past been considered threefold:

1. Effect of time lapse. In normal forgetting the remoter the event, the less well it is remembered. In RA—either due to progressive disease (for example, senility) or to trauma—it is held that the most recent events are the most severely affected.

2. Effect of context. In normal forgetting, important events often stand out and can be remembered when all else is forgotten. In RA due to organic disease and injury, amnesia tends to take little account of emotional bias. A man is just as likely to forget signing his will 2 sec before he was knocked out as to forget pulling up the chair to do so. When islands of memory are retained, they are typically of trivial visual images.

3. Irreversibility. Events which are forgotten in the normal course of time lapse can often be recalled with a little prompting. In true RA no form of assistance or priming appears capable of arousing an event

that has been blotted out. Even Pentothal narcosis has been tried (Russell & Nathan, 1946), and the difference between amnesias that can and cannot be reversed under narco-analysis has been held to distinguish the truly organic from the hysterical conditions.

However, is the difference between RA and normal forgetting really so great? The purpose of this paper is to analyze the situation more closely by considering first some of the published literature and then describing two further investigations.

Effect of time lapse: The temporal relationship between RA and trauma is probably not quite so rigid as has been generally believed. Analysis of residual RA's after head injury has shown that there are very frequently "islands" of amnesia extending beyond the immediate memory gap (Williams & Zangwill, 1952). Moreover, RA following electronconvulsive therapy (ECT), akin in nearly all respects to that following head injury, is not a completely all-or-none affair. Some effects of an experience – for example, a verbal habit set up by it – may remain even when the experience itself has been forgotten (Williams, 1950). In the much more extensive RA's following illnesses such as tuberculous meningitis (TBM), islands of memory also may occur or single impressions may be retained when the context in which they were experienced is forgotten. For example, a patient who made a good recovery after a long illness still retained an RA of about one year's duration four years later. During this year he had been in the army and had been sent on a clerical course. He had no recollection of his life in the army, but when shown a photo of his companions on the course, he was able to name each one. These islands tend to concentrate toward the remoter events – that is, the time furthest away from the onset of the illness – but a strict temporal relationship is lacking (Williams & Smith, 1954).

Equally, in normal forgetting it has been recognized ever since the work of Ebbinghaus that more is forgotten in the first few minutes after an event than at any subsequent time. The typical Ebbinghaus "curve of forgetting" shows that the amount remembered drops steeply in the first few seconds after an experience and thereafter levels out. Hence, what still can be remembered after 10 min is less likely to be forgotten in the subsequent hour than all that can be remembered after 10 sec. One would expect, therefore, if the effects of cerebral trauma paralleled or heightened the effect of normal forgetting, exactly the sort of temporal relationship in RA that is found.

Effect of context: Although all events falling within a few seconds of a concussional head injury or ECT or the onset of a severe illness may be difficult to recall spontaneously, this does not mean that they are wiped out completely. Some aspects often can be recalled with additional prompting, as was already mentioned (Williams, 1953), and, as with normal people, especially important events may be recovered more readily than others.

Irreversibility: This has never been considered to apply to more than the final state of RA. All observers have noted that RA shrinks in the early days of recovery and that shrinkage tends to be helped by cues, prompts, and revisits to familiar places. The exact point at which shrinkage stops is hard to ascertain. Some patients, after TBM with an RA of 2-3 years, may continue to recapture odd incidents as a result of conversations or visits for many years following their recovery (Williams & Smith, 1954). In rats the RA following ECT also shows evidence of alteration with time. Thus apparently "forgotten" behavior may return if the animal is returned to the situation where the behavior was acquired 7-10 days after a single shock (Zinkin, 1967). Failure to reawaken a memory by one means (for example, narcosis) does not necessarily mean that the memory may not come back in some other situation.

There is already evidence suggesting that the difference between RA and normal forgetting may not be so great as previously supposed. The following two studies further underline this suggestion.

I. RA after Head Injury and the Normal Retention of Daily Events

To find out how normal subjects retain simple daily events, four groups of nonbrain-injured hospital patients were interviewed.

Group 1 consisted of patients admitted for illness of sudden onset (perforated ulcers, fractures, etc.). Group 2 consisted of patients admitted for illnesses of gradual onset (for example, anemia).

Half of the patients in each group were interviewed the day after admission so as to be comparable with head-injury patients suffering short RA and posttraumatic amnesia (PTA); the other half were interviewed 2-3 weeks after admission to be comparable with the more severe head-injury patients.

Each subject was asked to describe

1. How he had spent the previous 24 hr
2. How he had spent the last 2 hr before admission to hospital
3. How he had spent some important day in his remote past, for example, his wedding day

It was noted that there was a big difference between the subjects' accounts of recent and remote events and of those preceding sudden trauma or gradual illnesses. Recent events and those preceding sudden trauma were recounted as sequences of isolated acts in which equal weighting was given to all events—"At 8:30 the alarm went and I made a cup of tea. The post arrived at 8:45. Then I let the dog out . . . ." More remote past events were organized into meaningful and coherent schemata in which individual events were often dropped out and could only be "supposed" or "guessed at" in recall.

It is concluded that unless a comparatively routine and unimportant event is followed by some catastrophe that causes its rehearsal, it becomes schematized and confused with other past memories. In RA following head injury the same pattern is seen after time lapse alone, but it appears that things that would normally be forgotten in a matter of weeks are forgotten after a head injury in days or even hours.

II. RA after ECT Compared with Normal Forgetting

Eighteen patients having ECT were asked to learn three pairs of words differing in meaningful association. They were given ECT 11-17 min after learning, and recall was tested 3 hr later.

The word lists were as follows:

SX = highly associated—e.g., score-cricket
SMZ = associated with mediator—e.g., Eden-(garden)-flower
SZ = not associated—e.g., angle-drink

The 18 ECT patients (Group 1) were compared with 10 matched controls (Group 3). In addition, 9 of the patients in Group 1 were retested with parallel word lists 3 weeks after the termination of treatment (Group 2).

In all groups of patients tested here, the SX lists were remembered better than the SZ, with the SMZ in between (Fig. 1). The differences between Group 1 and Groups 2 or 3 were the same on all lists, showing that the effect of ECT was similar to that of time lapse, only more intense.

III. Discussion

The above data have been quoted in support of the hypothesis that time lapse and cerebral disorganization have parallel effects on the recall of past events, that cerebral disorganization merely accentuates or increases the difficulty of retrieval of past experiences noted after

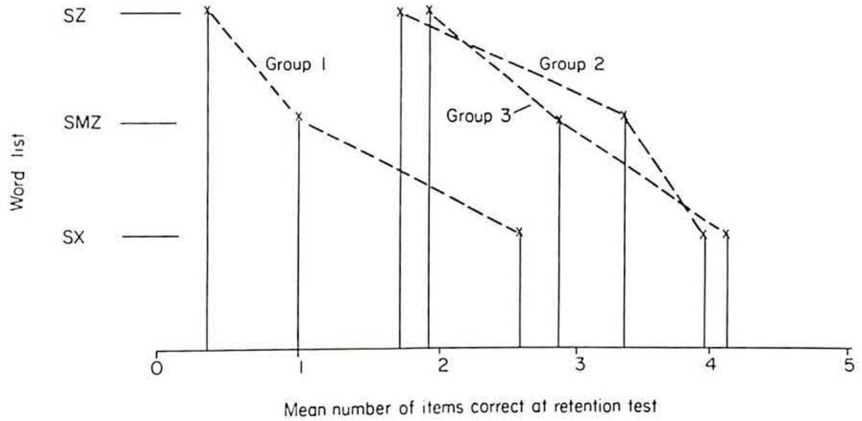

Fig. 1. Mean number of correct associations per group for three different lists.

time lapse. Such "forgetting" can be explained quite well by the model put forward by Weiskrantz (1966) and discussed in this volume by Zangwill. But what about the very short RA (2–5 sec duration) which seems to follow concussional head injuries marking them out so clearly from the head injuries, such as gunshot wounds, which do not cause loss of consciousness? For in contrast to the concussed patient who can very seldom remember the moment of impact, the patient with a nonconcussional head injury can usually describe in the same vivid detail as the man who breaks his leg all the circumstances of his accident. The difference in the two forms of description is undeniable —but is this difference due to the fact that consciousness has temporarily been disrupted, or is it due to the nature of the cerebral disorder which causes the disruption?

It has frequently been remarked, and is stressed further by others in this volume, that RA is never seen without some degree of posttraumatic amnesia (PTA), and the longer the PTA, the longer is the RA accompanying it (Russell, 1934), as a rule. This appears to have led to the assumption that some "mechanism" is involved which, if disrupted, causes not only loss of consciousness but also forgetting. But is the assumption of a special mechanism justified?

Again, it might be useful to compare the recall of incidents which *have* been followed by rehearsal (due to a subsequent catastrophe) and those which *have not*. When this is done it seems that events which are followed by some catastrophe tend to stick in the memory, whereas those which are not so followed drop out in the normal course of time. One patient who had broken his leg falling off a motor-cycle was able to describe in detail the antecedent events. "I always mend my own punctures," he explained, "and had just mended one that day. I suppose I must have bungled it, as going round a sharp corner the tire burst—and that was it." Asked when he had mended the puncture previous to this and what had happened subsequently, he looked quite offended and remarked, "How could I possibly remember that, nothing happened to fix it."

It seems that we are, in fact, forgetting an enormous number of simple, trivial events the whole time, and that the forgetting of these is more usual than their retention, unless something happens to make us rehearse and remember them. As a rule we never want to recall them, so do not appreciate their absence. It is only when we *do* want to recall them and find that their rehearsal has been prevented that we become aware of a blank. It is possibly this desire to recall rather than the absence of the memory itself which is the abnormality caused by the head injury. Thus, it is suggested that the short RA due to concussional head injuries is not due to any interruption of traces per se but only to an interruption of the rehearsal which might have led to their formation.

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POST-ECT AMNESIAS

Börje Cronholm

Since the introduction of electroconvulsive therapy (ECT), memory disturbances have been the subject of numerous investigations. The main purpose of this lecture is to review some studies, concerning post-ECT amnesia problems, performed in Sweden during the last decade by myself and my collaborators.

Memory disturbances after ECT may be classified into three categories. First, there is a total amnesia for the period of unconsciousness during treatment and for the period of profound confusion immediately following. Second, there is a retrograde amnesia, that is, difficulties in remembering events before treatment, especially what happened just before it, but also in recalling certain things that used to be well known. Third, there is an anterograde amnesia, that is, the patient may complain of difficulties in remembering recent occurrences, in learning new names, in recalling what has been read in the papers, and so on.

First I will report some studies of the anterograde amnesia after ECT. The aim was to analyze this disturbance in order to elucidate what specific aspects of the memory process may be involved and to construct a reliable method of measuring it. The initial hypothesis was that it is not so much the ability to acquire new knowledge but the ability to retain that which has been acquired that is the main characteristic of anterograde amnesia after ECT. To test this hypothesis I, with the assistance of the psychologist Lars Molander, developed tests whereby performance is studied immediately after a single learning session and 3 hr later, giving one "immediate memory score" and one "delayed memory score," the difference between those two scores being a "forgetting score." Various tests were tried; the final test battery consists of three tests, each containing 30 items. The first is a word-pair test, where there is some degree of relation between the two words but where the probability of guessing correctly is below 5%. In the second, a 30-figure test, the task is to remember pictures of 30 common objects and to point them out among a set of 30 others. (In some earlier studies a 20-figure test was used instead.) In the third, a 30-item personal-data test, six drawings of different persons are shown successively, and five pieces of information are given about each of them, for example, name, age, and education. Then the drawings are again shown, one at a time, and the subject is asked to tell what he remembers about each person. In studies where all three memory tests were applied, we formed combined memory scores by summing the raw scores. This was done on the assumption that all three tests—in spite of obvious differences as regards material to be memorized and the method of recall—at least to some extent measure the same underlying memory variables. An analysis of memory-score correlations in the three tests according to a device suggested by Campbell and Fiske has rendered some support to this assumption (Cronholm & Ottosson, 1963a). All three tests exist in two fairly parallel forms.

When memory performance before treatment is compared with that observed 6 hr after an electroconvulsive shock (ECS), a slight but significant decrease in immediate memory scores is accompanied by a far more pronounced increase in forgetting scores. The design of these experiments is shown in Fig. 1. The data presented in Table I are taken from a study by Ottosson (1960); they show memory performance after the first ECS in a series. Table II shows memory performance after the second ECS in a series (Cronholm & Molander, 1957); the influence on the forgetting score in the word-pair test seems to be even more pronounced. In this study we also compared performance on parallel forms of Benton's visual retention test, forward and backward digits, and cancellation of *d*'s in meaningful and meaningless text and in a multiple-choice vocabulary test. In none of these tests was there any decline after ECT—a striking contrast to the conspicuous increase of the forgetting scores. In both studies control groups also were examined. The control subjects received Hexobarbital but no ECS. Ottosson found a minor decline in immediate memory scores and a somewhat more pronounced increase in forgetting scores for the

Hours	First experiment, Form A or B	Second experiment, Form B or A
0	–	ECS
6	Presentation of material First recall: "Immediate memory score"	Presentation of material First recall: "Immediate memory score"
9	Second recall: "Delayed memory score"	Second recall: "Delayed memory score"

Fig. 1. Design of Anterograde Amnesia Studies.

control group—but far less than in the experimental group. I found no decline in memory performance in my control group.

The adverse influence on memory performance was found to persist 1 week after a series of 2–7 (with a mean of 3.6) ECS treatments (Cronholm & Blomquist, 1959). Applying the 30 word-pair and the 20 figure tests to a group of 30 patients, we found no significant decrease in the immediate memory scores but a significant increase of the forgetting scores (from 5.4 to 7.8) in the 30 word-pair test. One month after a series of 2–12 ECS treatments (with a mean of 5.3) we were not able to show any persisting negative effects in a group of 28 patients (Cronholm & Molander, 1964). In spite of this, memory disturbances of course may exist in some subjects even for a much longer time.

In the studies reported, the patients examined were chosen from among the patients at the psychiatric department of a general hospital. The prerequisite was that they should be easily testable and that their disease not be so severe that it could be assumed to have any noteworthy effect on performances in the tests.

However, most patients who receive ECT suffer from an endogenous depression with more or less pronounced cognitive inhibition as an important symptom. Complaints of "bad memory" are, in fact, very common among depressed patients. To study the influence of depression per se on memory performances, we compared a group of 20 inpatients, diagnosed as endogenous depression, with 20 nondepressed surgical patients (Cronholm & Ottosson, 1961). The depressed patients were paired off with the controls as regards sex, age, and level of

Table I
Anterograde Amnesia after ECS[a]

	Before ECS ($\bar{X}$)	6 hr after First ECS	$\bar{X}_D$	p
Immediate memory scores				
1. 30 word pairs	20.1	17.3	2.8	0.01
2. 30 figures	23.6	20.6	3.0	0.001
3. 30 personal data	16.8	15.2	1.6	0.05
Combined test	60.5	53.0	7.5	0.001
Forgetting scores (3 hr)				
1. 30 word pairs	4.7	10.6	−5.9	0.001
2. 30 figures	1.8	5.5	−3.7	0.001
3. 30 personal data	1.8	4.8	−3.0	0.001
Combined test	8.4	21.0	−12.6	0.001

[a] Siemens Konvulsator III. $N=30$. After Ottosson (1960).

Table II
Anterograde Amnesia after ECS[a]

	Before ECS ($\bar{X}$)	6 hr after Second ECS ($\bar{X}$)	$\bar{X}_D$	p
Immediate memory scores				
1. 30 word pairs	20.3	17.8	2.5	.001
2. 20 figures	17.3	15.8	1.4	.05
Forgetting scores (3 hr)				
1. 30 word pairs	5.9	14.0	−8.1	.001
2. 20 figures	1.3	5.7	−4.4	.001

[a] MacPhail-Strauss "Plexacon" Apparatus. $N = 24$. After Cronholm and Molander (1957).

education. As may be seen from Table III, immediate memory scores were significantly lower in the depression group whereas forgetting scores were very similar in the two groups. We also compared memory performances before and one week after four ECS treatments in a group of patients suffering from endogenous depression. We included only such patients who had benefited from ECT with respect to their depression. Immediate memory scores increased, indicating improved functioning in one aspect of memory; but forgetting scores also increased, indicating decline of functioning in another. A regression analysis indicated that there is a positive relationship between increase in immediate memory scores and rated improvement of depression but no relationship between change in forgetting scores and improvement. However, the results were not quite consistent among the three tests.

When discussing these findings, I will apply a simple model of underlying memory processes and make a distinction between three phases—registration, retention, and reactivation. I assume that both immediate and delayed memory scores are determined by all these processes but that retention is of greater importance for delayed than for immediate memory scores. Then the difference between these two scores—that is, the forgetting scores—would be mainly determined by retention. Thus, factors predominantly lowering the immediate memory scores would be assumed to have their main effects on the registration phase, whereas factors predominantly increasing the forgetting scores would be assumed to have their main effects on the retention phase. Using this frame of reference we may conclude that ECT has an adverse effect on retention and perhaps, but to a far less extent, on registration and reactivation. Of course, the increase of forgetting scores also may be due to impaired reactivation, but this would imply that the reactivation of memory traces 3 hours old is much more severely impaired than that of fresh memory traces. According to the rule of parsimony I thus prefer to assume an influence on retention. We also may conclude that memory impairment in endogenous depression mainly is due to an impairment of registration, whereas retention is not affected.

Retrograde amnesia after ECT comprises various phenomena. The forgetting of pathological experiences is common not only after ECT

Table III
Influence of Endogenous Depression on Memory Performance[a]

	Controls ($\bar{X}$)	Endogenous depression patients ($\bar{X}$)	$\bar{X}_D$	p
Immediate memory scores				
1. 30 word pairs	19.1	15.8	3.4	0.05
2. 20 figures	16.9	15.0	1.8	0.05
3. 30 personal data	17.4	11.8	5.6	0.001
Combined test	53.3	42.6	10.7	0.01
Forgetting scores				
1. 30 word pairs	5.2	5.6	−0.4	–
2. 20 figures	1.8	2.4	−0.5	–
3. 30 personal data	2.5	2.0	0.4	–
Combined test	9.5	10.0	−0.5	–

[a] Siemens Konvulsator III, three modifications. $N = 20$ in each group. After Cronholm and Ottosson (1961).

but also after spontaneous recovery. Furthermore, it is possible that the mechanisms behind patchy loss of old memories and the difficulty or inability to remember what occurred just before the treatment are not identical. The studies I will report have been restricted to the latter phenomenon; they concern remembering of material presented 1 hr or just before an ECS.

In several studies we used the same three memory tests as in the anterograde amnesia studies but with presentation and first recall—the basis of immediate memory scores—occurring 1 hr before the first ECS in a series, and the second recall—giving delayed memory scores—occurring 6 hrs after presentation, that is, 5 hrs after the ECS (see Fig. 2). Memory scores are compared with those obtained under control conditions on the day before ECS. As had been expected, we found a considerable increase in forgetting scores when an ECS was given 1 hr after presentation and first recall, while the administration of Hexobarbital, used as a control, had no such effect. In another control group, where there was no medical interference between the two testings, there was again no difference between the two occasions (Cronholm & Molander, 1961).

In another study we examined at what time a number (1-12 and 14-15) that the subject was asked to remember 5, 15, or 60 sec before an ECS actually could be recalled after treatment (Cronholm & Lagergren, 1959). The asymptote was reached 2-3 hr after the ECS; only a very few patients recalled the number later than that. In groups with the diagnoses psychoneurosis and endogenous depression, about 85% in the 60-sec group, 70% in the 15-sec group, and 60% in the 5-sec group finally recalled the number to be remembered. The recovery curve was steepest in the 60-sec group (see Fig. 3).

Hours	First experiment, Form A or B	Second experiment, Form B or A
0	Presentation of material First recall: "Immediate memory score"	Presentation of material First recall: "Immediate memory score"
1	–	ECS
|		
|		
|		
|		
6	Second recall: "Delayed memory score"	Second recall: "Delayed memory score"

Fig. 2. Design of Retrograde Amnesia Studies.

The studies of retrograde amnesia are in line with the assumption that after registration there is a period of fixation or consolidation and that this period is especially sensitive to disturbing influences.

Several studies have been devoted to an elucidation of factors possibly related to the adverse effect of ECT on memory functions. An important contribution was made by Ottosson (1960), who examined the importance of the cerebral seizure and other effects of the current on the anterograde amnesia. He compared four groups of patients. The general design may be seen in Fig. 1. In one group (A) the seizure was elicited by a stimulus considerably above the threshold for a grand mal seizure (supraliminal); in another (B), as in the routine procedure, by a stimulus only moderately above threshold; in a third group (C), by a stimulus as in the preceding method (B) but modified by lidocaine, administered intravenously. Lidocaine produces a considerable abbreviation of the EEG-recorded seizure and a modification of the seizure pattern. The fourth group (D) served as a control group and did not receive ECT. The results are summarized in Table IV. For the sake of clarity, only combined test scores are reported. As may be seen, the three treatment groups differ somewhat as regards decline of immediate memory scores, the decline being most pronounced in Group A and least pronounced in Group C. The same trend is still more evident as regards forgetting scores, and the difference between Group A and B is significant. These findings indicate that the seizure in itself has an adverse influence on memory functions but that other effects are still more important. This fact is especially interesting when related to another study in which a rating scale was applied on patients suffering from endogenous depression, before and 1 week after a series of four ECS treatments (Cronholm & Ottosson, 1960). The therapeutic efficiency was conspicuously and significantly less in the group where the cerebral seizure was shortened by lidocaine but approximately the same with supraliminal stimuli and in the routine procedure. Thus, the therapeutic effect in endogenous depression and the memory disturbances after ECT are not simply two manifestations of one and the same effect of the current on the central nervous system.

Retrograde amnesia has been compared in three other groups via the design already described (Cronholm & Ottosson, 1963b). In one group (A) seizures were induced with the Elther apparatus type ES; in another (B), with the Siemens Konvulsator III; and in the third (C), with that same apparatus but with the seizures abbreviated and modified by Lidocaine. With the Elther apparatus the amount of electrical energy necessary for eliciting a grand mal seizure is much less than

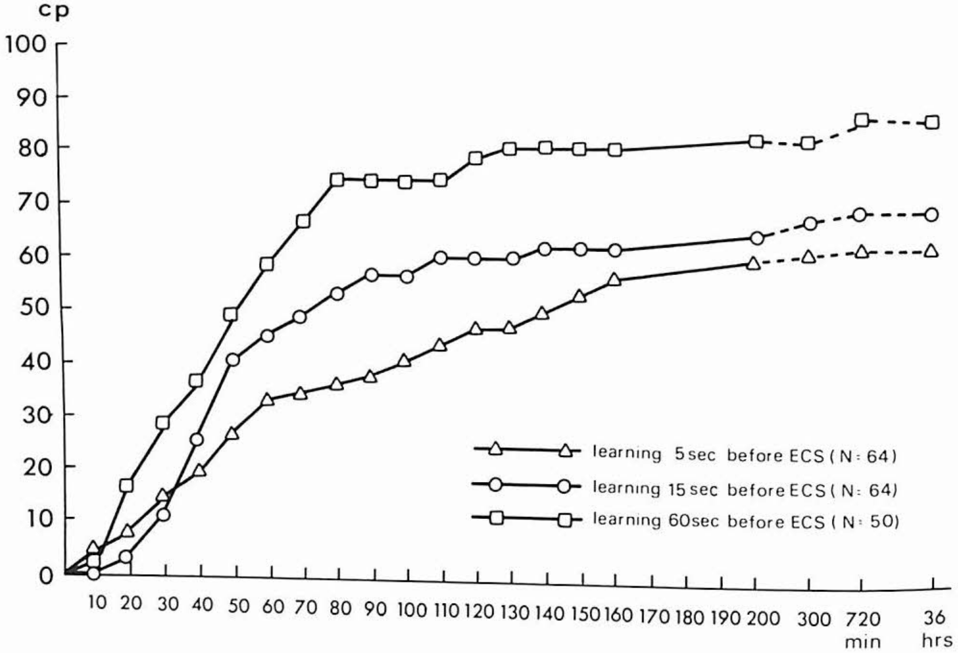

Fig. 3. Cumulative percentages of patients recalling a number learned before ECS as a function of time after ECS. After Cronholm and Lagergren (1959).

Table IV
Anterograde Amnesia after Various Modifications of ECT[a]

	A Supraliminal stimulation ($\overline{X}_D$)	B Routine procedure ($\overline{X}_D$)	C Seizure abbreviated and modified by lidocaine ($\overline{X}_D$)	D No ECT ($\overline{X}_D$)
Immediate memory score combined	8.4	7.5	5.6	−2.0
Forgetting score (3 hr) combined	17.7	12.6	10.0	5.5

[a] The table gives impairment of performance; improvement is denoted by a negative sign. Siemens Konvulsator III. $N = 30$ in each group. After Ottosson (1960).

with the Siemens Konvulsator. As may be seen in Table V, forgetting was least pronounced in the Elther group but approximately the same in the two others. This indicates that effects of the current other than the seizure are of paramount importance for the retrograde amnesia after ECT just as for the anterograde amnesia.

To sum up, after an ECS there is a retrograde amnesia for material

Table V

Retrograde Amnesia after Various Modifications of ECT[a]

	A Elther apparatus ($\overline{X}_D$)	B Konvulsator III ($\overline{X}_D$)	C Konvulsator III + Lidocaine ($\overline{X}_D$)
Forgetting score combined test	13.3	20.3	21.1

[a] $N = 24$ in each group. After Cronholm and Ottosson (1963b).

perceived shortly before the onset of treatment. This result may be due to greater sensitivity of memory traces during a period of fixation or consolidation. There also is an anterograde amnesia, which may be described mainly as a defect of retention. Further analysis of post-ECT amnesias would be of great interest but—unfortunately from a researcher's point of view—the number of testable patients given ECT has decreased during recent years due to the progress of psychopharmacology.

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THE ANATOMY OF MEMORY MECHANISMS IN THE HUMAN BRAIN

Raymond D. Adams

The title of this presentation is both inaccurate and presumptuous, for the work "anatomy" actually refers to a branch of biological science and the word "memory" to an abstraction denoting a feature of both learning and memorizing as well as other mental operations. It would be more correct to speak of the neural substratum of memory mechanisms. Nevertheless, the words indicate the general scope of this chapter and have the advantage of brevity.

The means by which the human nervous system is modified as a consequence of previous activity or, to come closer to the subject of this symposium, the way in which learned experiences are fixed, retained, and consciously recalled at a later time, can be approached from a number of standpoints. The problem can be regarded as one in the anatomy of neurons and neuronal interactions. More pertinently, it can be considered in terms of the function, that is, physiology and chemistry, of the nerve cells. Another equally valuable approach, because of the extreme complexity of the process, is a study in terms of psychology, that is, the relationships of the minute-by-minute life experiences of the human organism viewed either in terms of inner psychic function or behavior. Each approach is legitimate, and the proof of the usefulness and importance of them all is that they represent many different professional activities.The goal of all is to comprehend fully the neural basis of learning and memory and the causes of failure of these processes in the course of disease. Any complete theory ultimately must be reduced to the function of the nerve cell or systems of cells and be couched in the language of cytology, neurophysiology, and psychology.

The attitude of the scientific world toward the possibility of there being a determinable neuroanatomical basis of learning and memory has changed repeatedly during the past century. With the demise of the facultative or structuralist schools of psychology toward the end of the nineteenth century, the idea was widely held that all mental functions such as attention, perception, imagination, thinking, memory, affect, and drive were different aspects of a single, complex, and indivisible process. The research of certain eminent psychologists such as Lashley, which suggested the relative equipotentiality of cerebral cortex for certain of these more complex functions, supported this view. But psychologists investigating human intelligence were disturbed by the finding of a relatively high degree of independence, that is, poor correlation, among measures of memory and other cognitive functions such as capacity for coherent thinking, verbal skill, and arithmetic calculation. Another even more telling fact was the observations of neuropathologists who from time to time uncovered singular examples of disease in which an amnestic syndrome had exceeded in prominence all other clinical manifestations of cerebral disorder. The clinicopathologic study of these latter cases has provided a strong stimulus to the renewed investigation by experimental psychologists of learning and memory in primate behavior after local cerebral ablations. Thus has emerged more insistently than ever the idea that memory might be an independent function with its own chemistry, physiology, and anatomy.

I. Available Past Records for the Neuropathological Documentation of an Anatomical Substratum of Memory

A useful beginning here would be to refer to the review of Ojemann (1964), who collected all available cases in which a definable cerebral lesion had been associated with a unique defect in retentive memory. From this article it becomes evident that bilateral lesions of the medial parts of the temporal lobes (which include hippocampi, amygdaloid nuclei, and parahippocampal convolutions and connections with diencephalon via fornix and lateral forebrain bundle, the opposite temporal lobe via anterior commissure, the orbital frontal lobe, and other parts of the cerebral cortex), whether due to surgical ablation, infection, or infarction, have caused the most consistent effects on memory. In contrast, unilateral lesions of the same magnitude resulted in only mild and temporary deficits. Still unsettled is the question of whether lesions in the left (dominant) hemisphere interfere slightly more with retentive memory than do those of the right one. There is some evidence that left-sided lesions affect predominantly verbal (auditory) learning and memorization of verbal material, whereas right-sided ones impair learning of visual pattern discriminations.

In contrast, lesions in the frontal lateral parts of the temporal and parietal lobes, the cingulum, and the corpus callosum have been followed by little or no significant reduction in memory. However, the extent of lesions in which memory functions were studied has varied, and it is probable that all large defects in neocortical tissue may result in some quantitative reduction of memory.

Unfortunately, these observations usually are based on macroscopic examination and provide no more than a rough sketch of the topography of diseases that occasion unusually severe effects on learning and memory to the relative exclusion of other psychological functions. Nevertheless, they have led to the recent postulation that the hippocampal-fornical-mammillary systems of neurons constitute circuits essential to human learning and memory.

II. Types of Disease that Have Yielded the Most Promising Material for the Study of the Anatomical Problem of Memory

Ideally, diseases of brief duration with irreversible effects and restricted anatomical localization would be expected to offer the best examples for study. Diffuse or generalized diseases associated with many physiological and psychological effects are currently uninterpretable. Diseases of long, irregular, and unpredictable course tend to be unsatisfactory for the reason that one has no assurance of the state of pathology at the time of the clinical assessment. Finally, the disease should be one that results in a lesion that is easily visible under the light microscope, that is, has a definable pathology that is relatively irreversible.

Wernicke-Korsakoff disease, presumably a thiamine deficiency state, has provided the largest number of clinical examples. This nutritional-metabolic disorder, found usually in the alcoholic population of western European and North American society, develops within a few days at a certain stage of vitamin deficiency and is arrested by adequate diet on supplements of vitamin B_1. The lesion is clearly visible and has a constant and restricted localization in the walls of the third ventricle and periaqueductal regions and floor of fourth ventricle. The functional and structural changes are congruent.

Vascular lesions involving the medial parts of the temporal lobes, viz., infarct necrosis due to occlusion of branches of the posterior cerebral arteries (the two divisions of the basilar artery), have proved to be another rich source of material. Here the pathologic change occurs abruptly within an hour or so of the occlusion; the lesion is well demarcated. The deficit of nervous function in the form of a stroke is maximal in the first minutes or hours and regresses over the next six months to an irreversible residuum.

Acute inclusion body encephalitis, due in most if not in all instances to the herpes simplex virus and ranking as one of the most frequent and serious forms of viral infection in western Europe and North America, has consistently left in its wake a disorder of memory that exceeds all other functional cerebral deficits. The virus damages the orbital parts of frontal and medial parts of temporal lobes, sparing most other parts of the brain in the majority of cases. The topography appears to be explained in large measure by the proclivity of the virus to trek along the olfactory nerves to the parts of the brain in which they terminate.

Birth injury and various encephaloclastic processes of the paranatal and infantile periods often induce destructive lesions of variable magnitude in one or both temporal lobes. They are of interest here for the reason that they may give rise to temporal lobe epilepsy. Attempts at surgical therapy by ablation of one temporal lobe has on numerous occasions left the patient with a severe amnestic syndrome because of the coexistence of an occult lesion in the other side of the brain in the remaining temporal lobe. This sequence of events has accounted for a majority of the cases of incapacitating amnestic syndrome reported from centers devoted to the study of epilepsy.

III. The Clinico-Anatomical Method as a Means of Determining the Anatomical Basis of Disordered Nervous Function

The method to which allusion is made has been employed by many of the leading students of neurology since the time of Charcot. It involves the study of a neurological deficit in a diseased human being and then the anatomical study of his brain at the time of his demise. This method has yielded some of the most important ideas about the structure and function of the human brain. If the data to be obtained are to be granted the status of facts for medical science and theory, the method must be applied with scrupulous care as to detail. Otherwise there is chance of serious error. The method involves the following steps:

1. Definition and Measurement of the Disordered Nervous Function in Terms of Psychology and Physiology. This first step presupposes a sufficient knowledge of nervous activity to identify reliably some particular aberration of function. Furthermore, it should be possible to distinguish the negative, those due to deficits in function, from positive symptoms, those due to release of the function. Preferably the disorder should be one that expresses itself in the behavior of the patient, thereby being subject to accurate observation, but the patient's observations and estimate (introspection) of his own deficit also may prove valuable. The functional disorder should be quantifiable and expressible in terms of physiological psychology, which necessitates reference to the capacities of normal individuals of the same age. The last clinical observations should have been made shortly before death.
2. Complete Neuroanatomical Study by Serial Sections of the Whole Brain. Since the characterization of the lesion is relatively unimportant, its topography is best exposed by preparation of 25-μ thick celloidin sections, stained for nerve cells (Nissl) and myelinated fibers (Loyez myelin technique), taken at intervals of every 0.25–1.0 mm. This method divulges both the limits of the lesion(s) and the status of other noninvolved parts of the nervous system. Quantitation, which again is necessary if correlation is to be precise, entails comparison with standards obtained from the study of brains of normal individuals of the same age prepared in the same fashion.
3. Comparison of Several Cases of the Same Disease with Varying Symptomatology and of Two or More Diseases with Similar Symptomatology (The Study of Variance and Identity of Topography). Since no two nervous diseases have absolutely identical symptomatology, comparison of those cases manifesting a given "negative" symptom with those lacking it enables one to determine the responsible parts of the nervous system. Furthermore, since diseases of different types rarely exhibit a common topography, should a given "negative" symptom be common to two diseases it then can be related to that part of the neuroanatomical abnormality they share. This final step utilizes an

analytical method that might be designated as a study of covariance and coidentity of anatomical localization.

In contemplating the study of the anatomy of memory by this clinico-anatomical method, the difficulties become obvious enough. Plainly there is not one function that we call memory but several. The measurement of the most complex forms of human memory offer special problems to the psychologist because they involve introspection and report; hence ways must be found of ascertaining the reliability of memories of past experiences (the patient's statements must be checked). Testing the capacity for forming new memories (anterograde memory), a process equivalent to learning, involves many activities from the simplest types of conditioning to mastery of motor skills, language, mathematics, etc. Such a diversity of nervous functions obviously must depend on the activity of many parts of the nervous system. The separation of negative and positive symptoms in itself may seem relatively simple because psychiatrists seem generally agreed that all the "organic" type of amnesic state represents a deficit. But it would be a mistake to suppose that the nervous system of the patient with failing memory does not attempt some type of compensation. Probably confabulation, inactivity, lack of interest, avoidance of taxing situations, etc. are some of the secondary effects. Little is known about these latter states.

In the study of the anatomy of memory and learning disorders we are directed to parts of the nervous system that are poorly understood. For example, some of the thalamic nuclei and their connections with the medial parts of the temporal and orbital medial parts of frontal lobes in the human and primate brain have yet to be elucidated. Quantitation of neuronal and nerve fiber deficits in the diseased brain is hampered by lack of normative standards, which are just beginning to be assembled.

If one surveys all the reports of cases of cerebral disease in which there was a prominent amnestic defect of Korsakoff type, not a single one contains data that would satisfy the aforementioned requirements of adequate clinicopathological study.

IV. Nutritional Wernicke-Korsakoff Disease

Of the several diseases mentioned above that commonly affect the parts of the brain that are concerned primarily with learning and memory, the nutritional disease of Wernicke and Korsakoff most completely satisfies our criteria. Because of our interest in the neurological complications of chronic alcoholism, which has resulted in our having intimate contact with a large number of such cases, most of our observations of selective memory loss have been confined to them. In 1945–1950, when these studies were initiated, Victor and I were able to collect data on 219 alcoholics and, in addition, a few nonalcoholics, in all of whom the illness had developed on a background of severe, long-standing nutritional deficiency. We were able to satisfy ourselves by a number of systematic observations of the disease under the special conditions of full dietary control that the neurological disease may be prevented or arrested by the ingestion of thiamine chloride and that all other vitamins as well as proteins, fats, and carbohydrates have no effect on it.

The clinical syndrome was noted, usually, to evolve over a period of days and in its complete form comprised a bilateral ocular (abducens) and lateral gaze palsy with nystagmus, ataxia of gait, and either delirium or global mental confusion with apathy. A polyneuropathy of sensorimotor type in legs and sometimes arms, found in more than half the patients, completed the picture. As might be expected, every patient did not have the full syndrome. Polyneuropathy and mental symptoms were absent in about 20% of patients. Delirium tremens, a nonnutritional nervous disorder occasioned by the withdrawal of alcohol after habituation, complicated the illness in approximately a third of the patients.

The administration of thiamine chloride, even the small amount contained in a few balanced meals, led to reversal of the ophthalmoplegia, an improvement in gait, and a lessening of the apathy and confusion. Then emerged the clinical psychiatric syndrome known as Korsakoff's psychosis. The polyneuropathy slowly corrected itself over months, but the relationship of any improvement in it or in the Korsakoff's psychosis to thiamine remains unproven.

It was recognized almost from the beginning of our studies that this disease afforded a singular opportunity to study the amnestic syndrome because the underlying pathologic process has a remarkably predictable course; the pathogenesis appears to involve a subtle metabolic disturbance of certain systems of functionally related neurons leading finally to an obvious morphological change; and the lesions are usually distributed in a consistent pattern.

Concerning the relationship of the Korsakoff's psychosis to Wernicke's disease, our biochemical, nutritional, clinical, and pathological studies enable us to confirm the suggestion of Gudden (1891) that Korsakoff's psychosis is no more nor less than the disturbance of psychological function caused by Wernicke's disease, a fact that had been overlooked by both Wernicke and Korsakoff. In patients reaching the hospital with only opthalmoplegia, ataxia, and polyneuropathy but no mental disturbances, we were able to prevent both the early global confusion and the later Korsakoff psychosis—the latter by giving thiamine.

By serial examination of a group of patients with Wernicke's disease it was possible to follow the progression and regression of the psychic abnormality in detail and to analyze it. Always in its most acute and florid form there was obtundation of consciousness, diminution in alertness, restricted range of attention, imperceptivity, and confusion. Drowsiness occurred occasionally but coma, reported first by Wernicke, was present in only two of the 219 patients. During the confusion the patient would take little notice of his own condition or whereabouts and was totally disoriented and incapable of thinking, that is, analyzing problems. Often he misidentified people. It was only when these symptoms regressed under adequate diet with or without supplemental vitamin therapy that the full-blown Korsakoff psychosis with its characteristic loss of retentive memory and confabulation could be reliably detected. With further improvement the confabulation often disappeared and there was left a lesser degree of amnesia as a permanent residual state. In 60-75% of patients these latter symptoms were of such severity as to prevent return to a useful, independent life in the community.

A more detailed examination of the Korsakoff's psychosis, undertaken with Talland (1965), whose special observations on six of our cases have been presented in monographic form, revealed the following positive as well as the negative aspects of this syndrome. The positive features were identified as follows:

1. An inability to form new memories, that is, anterograde amnesia (prolonged practice does not help)
2. A loss of past memories, which had been formed before the beginning of the illness, that is, retrograde amnesia
3. Confabulation, that is, fabrications about past events in response to questions or in spontaneous conversation
4. Paucity of information and meagerness of content in conversation
5. Loss of insight, that is, lack of or incomplete awareness of the memory defect
6. Apathy, indifference, and incapacity to adopt a set or to persevere in complex activities

The negative features of the syndrome were identified as follows:

1. Preservation of normal capacity to think, solve problems, etc.; in the end stages of the disease intelligence test scores are probably

little if at all reduced from premorbid level despite gross memory defects

2. Preservation of an alert, attentive, wide-awake state of mind
3. Ability to perceive, imagine, speak, and calculate with customary speed and efficiency
4. Adequate cooperation and motivation in test procedures
5. Correct deportment
6. Lack of other neurological signs of cerebral deficit (EEG and CSF normal)

The intensity of the deficit was noted to vary from case to case. In some cases the patient remained unable to form any new memories over a period of 20-30 years and when asked about the past would act and speak as though the period never transpired. Others, mildly affected, admitted to a somewhat weak memory and could retain isolated events. A number of affected individuals were able to work under supervision. In all of them old memories of early life were more or less preserved (there are no adequate data on effect of disease on remote memory), which accounts for the fact that they seemed to have no difficulty in carrying out the usual tasks of daily living, that is, dressing, using kitchen utensils and common tools, speaking correctly, and so on. Approximately 20% of the patients recovered over a period of 1-10 months.

In comparing the residual effects of alcoholic Wernicke's disease with the amnestic syndrome that followed encephalitis (case of G. Talland and R. Adams), certain differences are noted. In the alcoholic the early confusional state is profound and apathy is more marked. As noted by Talland, there is a paucity of speech and activity and seldom persistence in any action. The alcoholic patient's inability to adopt new sets conducive to effective learning may be a central feature of the learning difficulty, but we are more inclined to relate this latter disorder to the lesions that underlie the global confusional state that earlier had dominated the clinical picture.

V. The Pathological Anatomy of Wernicke-Korsakoff Disease

Although the brains of a number of cases of acute inclusion body encephalitis and embolic or atherosclerotic occlusive disease of posterior cerebral arteries have been available to us, the lesions always predominate in medial parts of the temporal lobes. The variability of the pathologic changes and often the occurrence of death before the indefinable, reversible physiological derangement has subsided makes them unreliable for anything but crude clinico-anatomical correlations.

The postmortem studies on which our ideas of the anatomy of memory are based were conducted on 72 cases of Wernicke's disease. Of these, 37 had died in the acute stages (first few weeks) of the disease (usually from infection, cirrhosis, carcinoma, etc.), and 35 after it had reached a chronic stable stage, an average time lapse of 3.2 years from onset. A preliminary report of our findings was made by Adams, Collins, and Victor (1961).

Macroscopic examination of the brain always proved to be relatively uninformative. A slight atrophy of frontal convolutions was noted in many, but this involved a highly subjective assessment. In only three cases was the weight of the brain below 1200 gm. That there is, nevertheless, some degree of atrophy in the brain of this chronic alcoholic population was revealed more clearly in the size of the lateral and third ventricles, the anterior parts of which exceeded normal standards of size in approximately half the cases. On frontal section through the mammillary bodies some degree of atrophy and unusual coloration (orange-brown) or grayness was noted in 75% of our cases. In only 12% fresh hemorrhages were detected, always in the more recent cases. Grayness or granularity of the tissue in the medial thalamus, the periaqueductal region, and the superior colliculi and in structures in the floor of the IV ventricle were less frequent, and even more so were fresh hemorrhages in these regions. Atrophy of the folia of the superior vermis of the cerebellum, exposed usually only by section in the sagittal plane, was observed in one-third of the cases.

Under the light microscope the lesions were of variable appearance according to their age. In reticular formations nerve cells tended to be affected less than medullated fibers; this was most obvious in the mammillary bodies. Macrophages containing red blood corpuscles or hemosiderin (depending on freshness of hemorrhage) were more frequent than would have been suggested by naked-eye examination but were still absent in more than half of the cases. Pleomorphic microgliacytes and histiocytes, hypercellularity of the walls of small vessels, and enlarged astrocytes were prominent—the usual glio-vascular reaction. The greatest degree of histiocyte-microgliacyte-vascular change occurred in the earlier cases and cellular or fibrous gliosis in the old lesions. In the medial dorsal and medial pulvinar lesions the nerve cell loss and gliosis were unusually marked. Axis cylinder damage was difficult to demonstrate in all of the lesions, and we could detect only secondary degeneration of varying degree even in the mammillo-thalamic and mammillo-tegmental tracts, consequent to the mammillary lesions.

In conceptualizing the pathological process we came to view it as an incomplete regional necrosis of tissue that affected myelin more than nerve cell.

The topography of the lesions proved more interesting, but more elusive, than the histological characterization. Our observations vis-à-vis its neuroanatomical aspects were made on 53 cases, in 15 of which the entire diencephalon had been prepared in serial sections in either frontal or sagittal plane and in two of which the entire brain was in serial sections. Except in the two latter cases representative regions of the cerebral cortex and white matter were reviewed in random blocks, and the brainstem and cerebellum were sampled at multiple levels. Attention was focused on the diencephalon, mesencephalon, and cerebrum because it seemed unlikely that the lesions underlying the cognitive disorder to which we have referred could be located elsewhere. A total of 26 thalamic nuclei, the caudate and lenticular nuclei, other extrapyramidal nuclei in the subthalamus and midbrain, and all the hypothalamic nuclei were identified and studied in each case.

Turning first to the parts not involved, no consistent lesion was found in the cerebral cortex including the hippocampi. In cases of cerebral atrophy only a mild neuronal loss, not demonstrated for certain, could be put in evidence despite the use of special techniques for cell, myelin, axis cylinder, and glial cells. Hyperplasia of protoplasmic astrocytes accompanied all cases of hepatic failure. Senile plaques and Alzheimer fibrillary changes were observed in only one case. The caudate, lenticular, subthalamic, and red nuclei were spared in all cases. No lesions were observed in the cerebral white matter in either cell or myelin stains. A particular search for the cortical atrophy of Morel, an acknowledged alcoholic disease, and the degeneration of corpus callosum of Marchiafava-Bignami yielded completely negative results.

The part of the diencephalon, viz., hypothalamus, most frequently involved was the mammillary body (100% of cases), and another structure affected almost as frequently was the medial dorsal nucleus and median posterior nucleus, not clearly separable anatomically (88% of cases). In lesser frequency followed the anterior thalamic nuclei, paracentral, parataenial, medial ventral, lateral dorsal, paraventricular, rhomboid, central medial, reuniens medial pulvinar, and habenular. The severity of lesions was maximal in mammillary and medial dorsal and medial pulvinar nuclei and those less often implicated always tended to be more lightly touched by the disease. The nuclei that were devastated by the disease all lay in medial position, that is, within 2-3 mm of the ependyma. In other words, their position vis-à-vis the third ventricle appeared to be more important in determining their susceptibility than was any other attribute.

In the hypothalamus, after the medial mammillary nuclei, it was the dorsomedian, ventromedian, paraventricular, and the medial and lateral preoptic that suffered the brunt of the disease, in this lessening order of frequency and severity. In the mammillary body one could see that the termination of the fornices and to a lesser extent the origin of the mammillo-thalamic tracts (Vicq d'Azyr) were damaged. Exceptionally a scattering of lesions could be observed along the fornices, even as high as the genu. The mammillary lesion could be seen to extend superiorly into the medial ventral and dorsal nuclei of the thalamus.

In the mesencephalon, usually quite separate from the posterior thalamus, lesions were observed in 33 of the 43 cases studied by adequate sections. They involved the central periaqueductal gray matter in 55%, the oculomotor nuclei to a slight degree in 55%, the inferior colliculi in 33%, the medial tegmental region in 37.5%, the superior central (paraventicular) region in 70%, and the locus coeruleus in 68%. Again, involvement appeared to be a function of distance of nucleus in question from the aqueduct of Sylvius.

The upper pons was studied in all 53 cases, but adequate sections were in hand in only 31 and lesions were found in 24. Again, the principal pathologic alteration was in periventricular gray matter medial to the locus coeruleus. The tegmentum proper was not affected. In the lower pons and medulla oblongata the lesions conformed to a roughly triangular configuration involving slightly the abducens nuclei and paraabducens regions, the dorsomotor nuclei of vagus, the nuclei of tractus solitarius, and more severely the medial vestibular and spinal vestibular.

The anterior vermis and anterior lobule of the cerebellum was the site of disease in ten cases, that is, one-third of those in which it had been sectioned in sagittal plane. The peripheral nerves were affected in eight of the ten cases in which sufficient tissue had been obtained. Four cases exhibited varying degrees of central pontine myelinolysis.

VI. Clinico-Anatomical Correlation

The relation of the several components of the clinical syndrome to the regional pathologic anatomy of the disease process can now be determined more precisely than heretofore possible. Already the apathetic confusional state, the defect in retentive memory and learning, the ophthalmoplegia, the nystagmus, and the ataxia of gait have been specified as more or less separable entities, each tending to follow a rather different clinical course as the pathologic process regresses under therapy.

Before beginning the detailed analysis, however, it should be pointed out that in every case in which the diagnosis of Korsakoff's psychosis was mentioned in the clinical record, Wernicke's disease was demonstrated at autopsy. This datum, along with the clinical, nutritional, and metabolic studies carried out in this laboratory as well as in others, corroborates the identity of the two conditions. Clinically it is of interest that the lesions of Wernicke's disease were found in three cases of pure Korsakoff psychosis or amnestic state even though the ophthalmoplegia and ataxia of Wernicke's disease had not been described.

The greatest uncertainty attaches to the anatomical basis of the apathetic-confusional state, which was observed in all of our 23 acute cases. Since in the more chronic cases this part of the syndrome tends to be reversed under therapy, later exposing the amnestic state, it surely does not depend on a visible structural change. One can only conjecture that the presence of acute disease in the diencephalic-mesencephalic region, near the reticular activating systems of cortical neurons, might explain a temporary physiological derangement of these latter structures. However, we have observed other cases in which the lesions of Wernicke's disease were not found, and we have no way of deciding whether the clinical observations were inaccurate or a delirium tremens or hepatic stupor were mistaken for early Wernicke's disease.

With reference to the amnestic-confabulatory complex we looked first to the hippocampal-fornico-mammillary system since other studies and evidence from other diseases had incriminated these parts. Actually lesions were found in the mammillary bodies in every case of Wernicke's disease in which there were adequate sections of this posterior part of the hypothalamus. However, in four cases that had no memory defect mammillary lesions were also demonstrated, and it was not a question of degree of involvement or recent onset of disease after the negative clinical examination, because the lesions were as severe as in others with an amnestic state and in three of the four were of chronic type. The dorsal hypothalamus was affected in only one of these cases, but since lesions in this region were of relatively low incidence in the amnestic cases, little significance could be attached to it.

The medial dorsal nuclei of the thalamus were the parts most definitely related to the memory abnormality. In the 26 cases with certain Korsakoff psychosis it was the only thalamic nucleus that was extensively involved. However, the pulvinar, geniculate bodies, and habenular nuclei were not completely surveyed. The latter two nuclei are not considered important in the topography of the disease, since they are so infrequently affected in a patient with memory loss. The pulvinar may be important; we had only one case with a medial dorsal thalamic lesion with sparing of pulvinar. In two patients with memory defect the mammillary bodies were minimally affected and the medial dorsal thalamic nuclei were more severely involved. The hippocampi and hippocampal convolutions and proximal fornices were normal or nearly so in all cases.

A word or two about the anatomical bases of the ophthalmoplegia, nystagmus, and cerebellar ataxia might be added. The horizontal nystagmus appeared to correlate with lesions in the lower brainstem, particularly in the medial and spinal vestibular nuclei, and vertical nystagmus possibly in the upper brainstem periaqueductal region. However, in five of the cases that had no residual nystagmus lesions were present in the vestibular nuclei. The ophthalmoplegia, being in all instances transitory, can be ascribed to lesions near the abducens and oculomotor nuclei, and the lateral gaze palsy to a physiological derangement of the lateral gaze center lateral to the abducens nuclei. Surprisingly, small lesions (nerve cell loss) could be present in both the abducens and oculomotor nuclei without residual signs of permanent gaze or ocular muscle palsy. In all cases of cerebellar ataxia in which the cerebellum was examined by sectioning in the sagittal plane, cortical atrophy of the superior vermis was demonstrable. But there were ten other cases with mild cerebellar ataxia where sections in horizontal plane did not pass through the superior vermis. Therefore, a final statement as to the anatomical basis of this symptom awaits the examination of further cases.

VII. Neuroanatomical Bases of the Amnestic Syndrome

Taking into account the anatomical data derived from the study of Wernicke-Korsakoff disease along with data obtained from the neuropathologic study of other diseases, one can summarize as follows:

1. Alcoholic Wernicke-Korsakoff syndrome is evidently based on lesions in the medial diencephalon, particularly the medial dorsal nuclei, and not in the mammillary bodies as claimed by Gudden (1891).

2. Atherosclerotic or embolic occlusion of posterior cerebral arteries causes the amnestic syndrome by bilateral infarction of the medial parts of the temporal lobes, particularly the hippocampal and parahippocampal convolutions and the fornices including psalterium (Glees & Griffith, 1952; Victor, Angevine, Mancall, & Fisher, 1961).

3. Surgical ablation leads to the amnestic syndrome if the medial and inferior parts of one or both temporal lobes are excised, or if one is excised in the presence of disease in the other (Scoville & Milner, 1957; Penfield & Milner, 1958; Walker, 1957; Falconer, 1955).

4. Acute inclusion body encephalitis causes an amnestic syndrome by inflammatory necrosis of medial parts of the temporal lobes (Rose & Symonds, 1960; Drachman & Adams, 1962; R. D. Adams & G. A. Talland, unpublished data).

Unfortunately, not a single human example of any one of these four diseases has been adequately studied by clinical and anatomical methods. Our own cases of Wernicke-Korsakoff's disease that were subjected to full anatomical study had not been seen by us nor subjected to proper psychological tests; and those with full clinical studies have not come to autopsy. Only in the case of Victor, Fisher, and Angevine, of atherosclerotic occlusion of posterior cerebral arteries, have reasonably full notes on the clinical state and pathological anatomy been obtained, and here the lesions extended beyond the hippocampal regions into the adjacent parahippocampal and fusiform regions. None of the surgical and inclusion body encephalitis cases has been fully studied anatomically. Thus one can see the need for a collaborative effort between clinicians and neuropathologists if our knowledge of the precise anatomy of the memory and learning processes is to be consolidated.

The discovery that any one of these disease processes will consistently produce a failure of the capacity to form new memories, combined with retrograde loss of memories that had presumably been formed before the illness began, has been of singular importance in the study of the physiology of man's cerebral mechanisms. Equally important is the recognition of special mechanisms subserving these functions in the medial temporal lobes and medial diencephalon. It must not be supposed from this that other parts of the neocortex do not play a part in mnemonic integration. Rather, it is in the medial portions of the temporal lobes and their connections with the medial diencephalon where the smallest lesions cause the most profound derangements of these functions. They therefore must represent essential circuits that act in relationship to the upper reticular formation and neocortex. Special learning and memorization processes for language, arithmetic calculation, and visual spatial orientation without question have a neocortical localization in lateral temporal and parietal lobes. One might say that the hippocampal-diencephalic mechanism is the universal circuit for learning and memory (U factor) and the neocortex the special ones (S factors)—to recast our concepts in a language reminiscent of that of Spearman for intelligence. This is only a hypothesis to be tested by further psychologic, physiologic, and pathologic studies. From the available pathologic data the common attribution of the mnemonic function to hippocampi, fornices, or mammillary bodies does not stand critical analysis.

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HYSTERICAL AMNESIA[1]

John C. Nemiah

It might be valuable for this discussion to review briefly and systematically the basic observations concerning hysterical amnesia and related phenomena, as well as one or two fundamental concepts relevant to them, although the reader is familiar with them to a greater or lesser degree.

I shall start with a short clinical vignette for which I am indebted to Dr. George Talland, who referred me to the article in which it is contained—a study in Italian by Romolo Righetti (1920). He presents the following case history:

> On the morning of the second of December, 1912, Joseph P., an unmarried travelling salesman of about 23 years of age, for personal reasons, fired a pistol shot at his sister-in-law, causing a slight wound in her lumbar region; then, suddenly turning the revolver on himself, he fired several shots point blank in the direction of his right auditory canal. As x-rays later confirmed, one bullet penetrated the cranial cavity through the petrous bone, injuring the brain substance of the right temporal pole, and the contiguous portions of the frontal lobe of the same side. As an immediate consequence of the wound, the patient fell face down on the ground, with a serious hemorrhage from his right auditory meatus, and lost complete consciousness for a period of about an hour. Taken to the hospital, the injured man, during the initial phase of the treatment, deliriously uttered a few words ("That whore!"), alluding to the lady at whom he had shot.

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[1] From the Psychiatric Service of the Massachusetts General Hospital; from the Massachusetts General Hospital Division of the Hall Mercer Hospital; and from the Department of Psychiatry, Harvard Medical School, Boston.

Let me summarize the sequel: The patient recovered; but for more than 2 years thereafter, systematically repeated testing of his mental functioning revealed a total loss of memory for a period that extended from a month before his homicidal-suicidal act to a date 40 days after the event. Suddenly, 2 years and 3 months later, all the particulars of the impulsive event returned to him in a dream and remained in consciousness when he awoke. Soon he recovered all his lost memory, except, as the case history concludes, "There persisted only a short gap in memory corresponding to the period of post-traumatic confusion."

Brief reflection on these observations shows us that two types of amnesia are manifested. (1) There is a small area of amnesia covering events of a short period of 2 or 3 hours in which there was an obvious, gross dysfunctioning of brain processes, accompanied by an alteration of consciousness. The events of this short period were totally and permanently beyond the patient's recall. (2) There was a second, larger body of events spreading over a period of 70 days, which was, for a limited period of time, totally beyond the patient's recall. This amnesia was not associated with obvious, gross brain dysfunction (especially that segment of it antedating the shooting), and ultimately the events of this period were completely recalled by the patient.

At the risk of oversimplifying, let me propose that what distinguishes the two forms of amnesia seen in this patient is the capacity to recall to consciousness memories of past events. A basic characteristic of hysterical amnesia is just this capacity ultimately to recall to consciousness memories of events that for a greater period of time were beyond the patient's power of recall and were thereby excluded from consciousness. This being the case, the different patterns of recall are an important determinant of the clinical types of amnesia. Let us briefly review these.

1. Simple Amnesia. This is exemplified by the patient I have cited and is the type we commonly see clinically today. The events of a circumscribed period of time (from a few moments to many days) are covered by an amnesia that likewise may persist for a few hours to many years.

2. Alternating Amnesia. Two separate states of consciousness alternate with one another, during each of which there is no memory for events that occurred during the other. Sidis and Goodhart (1905) provide us with a good example of this: A young clergyman, Mr. Hanna, following a carriage accident, developed a total amnesia for his entire previous life. (Let us call this his "A state.") For several months the amnesia persisted for the events antedating the accident, though he

could recall everything that occurred during the period following the accident. (Let us call this his "B state.") One morning he awoke with a total amnesia for the events of his newly developed B state but with full memory of his A state. Several weeks later, his morning awakening found him once again in his B state with no memory of his previous two A states. Thereafter his clinical course was one of alternating states, each with amnesia for the other, so that eventually in either he could not recall great chunks of his previous life. This was disconcerting to say the least, and it was particularly difficult for Mr. Hanna. In his B state he enjoyed the finer things of life, like smoking and drinking. On occasion he would awake in the morning in his A state after a hard night of pleasure in his B state, and his clergyman's conscience would be troubled by the foul aftertaste in his mouth of stale beer and old cigars.

3. The Multiple Personality. In a sense, Mr. Hanna was two different people alternately inhabiting the same body—a veritable Dr. Jekyll and Mr. Hyde. He represents a local stop on the way to the full-blown multiple personality. Here several different states of consciousness, each a complex set of memories, attitudes, and behaviors with a distinctive personality, is sequentially and disjointedly manifested by the person suffering from the disorder. Often, in one state of consciousness not that of the "normal," premorbid personality, the patient can recall all the events occurring during the other personality states, each of these others, however, having a total amnesia for all the rest. I have not time to give you a clinical example here, but I refer you to Morton Prince's (1906) classic description of his patient Sally Beauchamp.

One need not restrict oneself to the clinical problem of hysteria to find amnesia of the sort under discussion here. It is in the first place a phenomenon well known to probably everyone of us in the form of a universal occurrence. I am referring to the experience of forgetting a name or a word that you "know as well as you know your own name," as the saying goes. Try as you will, you cannot recall it until, giving up the struggle, when your mind is occupied with something else, the forgotten item "pops into your mind." Many of you no doubt have had a similar experience with dreams. Soon after one awakens, a long and rich dream is quite inaccessible to voluntary recall, but later in the day, in association with a conscious thought, sensation, or perception, part or all of the forgotten dream rises spontaneously into memory. The universally found amnesia for most of the events of early childhood is well known. On occasion long-forgotten childhood memories emerge into consciousness with a dazzling, often exciting clarity.

Proust has recorded such an event in *Swann's Way* in his description of the affair of the cup of tea and madeleine. It is too long to reproduce for you now, but it is one of the most striking and graphic examples I know of. Furthermore, hypnosis furnishes us with further relevant observations in the form of the *posthypnotic amnesia*. Here, following his awakening from the hypnotic state, the subject either spontaneously or on the command of the hypnotist is unable to recall the events that occurred under hypnosis but can bring them into consciousness when the hypnotist orders him to do so.

At this point, let me review with you three further salient characteristics of the amnesia under consideration:

1. Although the memories of events covered by the amnesia are totally inaccessible to voluntary recall, they nonetheless may influence the contents of the individual's conscious thoughts or his behavior, without, however, his necessarily recognizing the contents or their connection with the forgotten events. The content of dreams may be so determined, as in the case of the patient I described at the start. Mr. Hanna, during his B state, would have vivid dreams of people, places, and events. These were totally unfamiliar to him, but his parents recognized them as being representations of real occurrences during his A state. Occasionally a long-forgotten and previously unrecoverable event will emerge in the dream imagery of normal people. A striking example of this is found in Hammond's (1869) *Sleep and Its Derangement*:

> In the course of his (a friend's) practice as a lawyer, it became necessary for him to ascertain the exact age of a client, who was also his cousin. Their grandfather had been a rather eccentric personage, who had taken a great deal of notice of both his grandsons—his only direct descendants. He died when they were boys. My friend often told his cousin that if his grandfather were alive there would be no difficulty at getting at the desired information, and that he had a dim recollection of having seen a record kept by the old gentleman, and of there being some peculiarity about it which he could not recall. Several months elapsed, and he had given up the idea of attempting to discover the facts of which he had been in search, when, one night, he dreamed that his grandfather came to him and said: "You have been trying to find out when James was born; don't you recollect that one afternoon when we were fishing I read you some lines from an old text of Horace, and showed you how I had made a family record out of the work by inserting a number of blank leaves at the end? Now, as you know, I devised my library to the Rev. Arthur Smith. I was a damned fool for giving him books which he will never read! Get the Horace, and you will discover the exact hour at which James was born." In the morning all the particulars of this dream were fresh in my friend's memory. The reverend gentleman lived in a neighboring city; my friend took the first train, found the copy of Horace, and at the end the pages constituting the family record, exactly as had been described to him in the dream. By no effort of his memory, however, could he recollect the incidents of the fishing excursion.

Psychopathologic symptoms provide us with further observations of this phenomenon. A young lady was admitted to our psychiatric ward because of an amnesia covering a period of 12 hr. During her hospitalization she had a recurrent hysterical hallucinatory vision of a girl running through a full parking lot. The content of the vision was totally strange and foreign to her. When her amnesia was lifted and she remembered the occurrences of the lost 12 hr, this was found to be a central event in her actual experiences during that period. Another patient came to our ward because of acute anxiety over a recurrent, obsessional image of herself and her father in a naked sexual embrace. In the course of treatment she recollected, with near panic, and in a direct train of associations to the symptom, having seen her parents having sexual intercourse when she was 5 years old—a memory that had been totally beyond conscious recall for 15-20 years. (Incidentally, it is of interest to note that following the return of this memory, her obsessional symptom vanished.) Breuer (Breuer & Freud, 1955) describes in the *Studies on Hysteria* a young woman with a hysterical inability to drink water. The onset of the symptom was traced under hypnosis to the otherwise totally forgotten event of seeing with considerable disgust a dog drinking from the glass of a fellow employee in the household where she worked. The revival of the memory coincided with the disappearance of the hysterical symptom. Finally, I should mention the phenomenon of posthypnotic suggestion, in which a command given by the hypnotist during hypnosis is carried out by the subject after awakening with *no* memory of having been instructed to perform.

2. A second characteristic we should notice is the sense of mental effort that is experienced by the individual when he is attempting voluntarily to recall the lost memory. Proust describes this beautifully in the passage in *Swann's Way* I mentioned earlier, but you can probably corroborate it from your own experience of trying to remember a forgotten name; it is hard, unrewarding, and often unpleasant mental work. I also might remind you of the pleasant exhilaration that often accompanies the sudden spontaneous return of the lost memory. A correlate of this sense of effort is what is termed the *resistance* that the observer may detect when he is struggling to remove an amnesia: The patient becomes anxious, stops talking, changes the subject, and may even abruptly leave as he is pressed to recall what he has forgotten.

3. This leads us to a third and frequent characteristic of amnesia—the fact that the memories and events thus forgotten may be painful ones, producing anxiety, panic, sorrow, despair, or guilt if and when

they emerge into conscious awareness as a recollection of one's own experience and have meaning and significance. The young woman patient with amnesia mentioned a short while ago had forgotten a series of events that evoked a feeling of despairing loneliness and hopelessness, the ego-alien hallucinatory scene in the parking lot being a fragment of her desertion by her boy friend that immediately preceded the onset of her amnesia. The guilt, shame, and nearly overwhelming panic of the other patient with the obsessional image of her father was all too evident to the doctor as she recalled her childhood observation of her parents' sexual intercourse. Even in minor acts of forgetting a name, one can often discover that the forgotten item is intimately associated with a subject that is emotionally painful to the forgetter. I refer you to Freud's (1951) *Psychopathology of Everyday Life* for numerous examples of this.

My concluding task is to discuss briefly three basic concepts of relevance to the form of amnesia under discussion here. First there is the notion of *unconscious mental events*. In the patient who shot himself in the head we saw that we were dealing with two types of amnesia. The totally irreversible amnesia of 2 or 3 hr was related to the acute phase of his brain damage. In this, and in other cases of damage to the central nervous system, although we may not be able to define all the details of the mechanisms involved, we attribute the memory disturbance to the gross, detectable disruption of brain structure that either destroys the anatomical substrate of memories or prevents incoming sensations from being stored. There are no memories (and never will be) because there are no neuronal memory traces. Our concepts and explanations in this instance are *physiological* in nature. In the case of the patient's amnesia for the 70-day period the situation is different. Here the memories, though unavailable to voluntary recall, were potentially there for a return to consciousness under appropriate circumstances. Physiologically speaking, the memory traces were there, but for some reason the memories themselves could not become conscious. In this instance we speak of the memories as being unconscious, which is a *psychological concept*. This is not meant to imply that there are no physiological processes underlying the events but only that we do not know what they are and therefore resort to concepts and words that refer to mental, psychological events and experiences: At one point memories are absent from consciousness despite attempts to recall them voluntarily, and at a later time they appear, complete and detailed, in conscious awareness.

A concept closely related to that of unconscious mental events is the notion of *repression*. If it is not found in every instance of an unconscious mental event, it is often enough associated with them to form a significant subclass. "Repression," like "unconscious," is a psychological term and refers to a psychological force or process that actively keeps mental events from emerging into consciousness and directly counteracts the mental act of voluntary recall. The conscious experience of work, effort, and resistance when attempting to recall unconscious memories is a manifestation of repression.

Finally, we must consider the notion of *psychological defense*. This contains the idea that what is rendered unconscious by repression is repressed because it is potentially painful. Repression, therefore, serves the function of protecting or defending the individual against the mental pain (for example, anxiety, despair, and guilt) he would suffer if the memory were to emerge into conscious awareness. Again, not all repression may be defensive in character, and we may be dealing here with an even smaller subclass of amnesia, in which the amnesia is seen as a *clinical* manifestation of the psychological mechanism of repression and other symptoms (for example, hysterical hallucinations) are seen as the fragmentary and often distorted return of the repressed. As such it exemplifies the psychodynamic scheme of psychological conflict and psychological symptom formation that provides a useful theoretical framework for understanding clinical emotional disorders.

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II

EXPERIMENTALLY INDUCED DISORDERS

THE PLACE OF SIMPLE BIOCHEMICAL EXPERIMENTS IN THE STUDY OF THE MECHANISM OF MEMORY STORAGE[1]

Samuel H. Barondes

The overall process of learning and memory storage is highly complex. Even the simplest forms of learning involve the participation of large numbers of neurons and probably induce alterations in the relationships between at least a few of these. However, the complexities of memory storage are probably related to the complexities of neuronal circuitry in the brain. They do not preclude the possibility that a small number of relatively simple biological regulatory processes have evolved in the nervous system for the establishment of memory by the alteration of interneuronal relationships. The situation may be similar in many ways to that of biological differentiation. An understanding of the entire complex of processes that lead from ovum to man seems very difficult. Yet the principles that govern cellular replication, differentiation, and the ultimate development of relationships between cells may prove to be relatively simple. Likewise a detailed analysis of the entire process of learning and memory may prove very difficult, but the basic mechanisms that regulate the interneuronal relationships that mediate memory may be far simpler to discover.

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The purpose of this paper is to review some ideas about how interneuronal relationships may be governed and to discuss some experiments that illustrate the type of approach we have been using to evaluate this problem. The basic speculations that underlie this discussion are as follows:

1. That at least two types of biochemical change are employed for memory storage—a fairly rapidly reversible change that may mediate "short-term" memory and a relatively stable and possibly self-replicating change that may mediate "long-term" memory
2. That both these types of changes effect an alteration in synaptic transmission at some critical synapses in the nervous system
3. That "short-term" changes are mediated by structural changes in protein, whereas "long-term" changes are mediated by protein synthesis

These speculations are presented not only to verbalize the hope that the underlying mechanisms that mediate memory may prove simple but also because they have led to studies of possible biochemical mechanisms involved in memory storage as well as to studies of general brain protein metabolism that may prove relevant to behavioral investigations. I propose to review my own work in this field as an example of the relevance of this approach for the study of memory.

I. Forms of Memory Storage

There is evidence that memory is stored in at least two forms—a "short-term" form and a "long-term" form. Disruptive procedures like electroconvulsive shock (ECS) markedly impair memory if administered very shortly after training but not if administered long after training (McGaugh, 1966). This suggests that memory is in a labile, "short-term" form that is susceptible to ECS for some period after training but is later stored in a stable "long-term" form. There is, however, considerable uncertainty about what these findings imply with respect to the underlying mechanisms of memory storage, largely because the duration of the period of sensitivity to ECS varies markedly in different situations. In some experiments seizures must be induced within 5 or 10 sec after training to effect amnesia (Chorover & Schiller, 1965), whereas in other situations seizures induced hours later (Kopp, Bohdanecky, & Jarvik, 1966) cause some impairment of memory storage. This variability precludes a simple, uniformly applicable and universal formulation about the temporal sequence of phases of memory storage. It does not, however, mitigate against the general notion that there are at least two separate forms of memory storage. Rather it indicates only that in some situations the long-term process may be effectively established more rapidly than in others. Nor do the various possible times of onset and termination of short-term and long-term memory storage processes provide us with any information about the underlying types of biochemical mechanisms. Many types of biochemical changes can be effected rapidly, and many of these might be either transient or enduring. Structural changes in proteins may occur instantaneously. Even the biosynthesis of whole protein molecules can occur in a matter of seconds. Reversal of such changes, whether based on structural alterations in proteins or on protein synthesis, may be rapid or slow. Examples of rapidly reversible structural changes include the "allosteric" processes in which transient alterations in protein configuration are produced. More slowly reversible structural changes are exemplified by the reversible incorporation of a single carbohydrate moiety into a protein. Irreversible structural changes may occur if a portion of the protein molecule is cleaved, thereby activating the remainder as in the case of conversion of proenzymes to enzymes.

The rate of reversal of changes based on protein synthesis also may be relatively rapid or slow. Some proteins probably are degraded within a matter of hours after their synthesis, as has been suggested for tryptophane pyrrolase. Others may not be degraded for months or years, as appears to be the case with collagen. Therefore, the implication of studies of temporal sequence of memory storage is that memory is probably stored in at least two forms – a long-term form and a short-term form, but no information is obtained from these studies about the types of biochemical processes that mediate these forms.

II. The Critical Role of the Synapse

It seems extremely likely that the process that mediates memory storage ultimately must effect an alteration in synaptic transmission somewhere in the nervous system. Although many arguments have been presented against the concept that there is strict determinism in all the specific synapses employed in the execution of any act (John, 1967), it seems very likely that at least a few of these synapses in the long chain that culminates in behavior are strictly determined. By facilitating transmission across one or a few critical synapses it is possible to regulate the occurrence of a certain behavior, the details of which may be mediated by a number of alternative neuronal chains. It is true that there may be a multitude of critical synapses in different parts of the brain that may regulate critical circuits for any given type of behavior, so that ablation of even large regions will not eliminate memory because of this property of redundancy. The synapse, however, remains the crucial site for regulation of neuronal activity, and it is to the synapse that one must look for possible answers as to how memory is stored.

III. Possible Mechanisms for "Transient" and "Permanent" Memory

There are many possible ways in which both transient (short-term) and permanent (long-term) changes in synaptic transmission can be effected. Changes in either the presynaptic or postsynaptic portion of the synapse could produce them. Although studies of the duration of phases of memory storage have not assisted us in uncovering the type of biochemical regulatory mechanism probably involved in these two forms of memory storage, it seems likely, from general considerations, that transient memories are mediated by structural changes in preexisting molecules whereas permanent changes are mediated by biosynthetic processes. This is of course completely speculative and is prompted only by the fact that structural changes in molecules tend to be fairly rapidly reversible, whereas changes induced by the biosynthesis of macromolecules tend to have a somewhat longer duration. Since there are exceptions to this generalization, however, this assertion must be viewed only as a loosely held hypothesis.

From a knowledge of a multitude of possible biological regulatory processes that may have been adapted in the nervous system, one can provide many examples of how short-term and long-term alterations can be brought about. Short-term alterations in synaptic efficiency could be produced by a structural change in proteins in the postsynaptic membrane that render it more permeable to transmitter. Such structural changes could be produced by the addition of sialic acid to a glycoprotein already present in the membrane and could be reversed by cleavage of the sialic acid residue from this glycoprotein. Long-term changes might be effected by the biosynthesis of protein that makes its way to either the presynaptic or postsynaptic portion of the synapse and alters its permeability to transmitter substances or its propensity to reaccumulate transmitters or to degrade transmitters.

These changes based on the synthesis of protein might, because of the altered intercellular relationships produced, feed back to the cells involved and cause further synthesis of similar materials. This might ensure replacement of the critical proteins which, like most proteins, would be expected to be susceptible to degradation.

The purpose of presenting these highly speculative considerations is to demonstrate why it seems reasonable to undertake relatively crude biochemical investigations of the highly refined and complex problem of memory storage. As examples of this approach I will describe two types of studies I have been engaged in, one analyzing the effects of inhibitors of cerebral protein synthesis on memory and the other concerned with the transport of proteins to nerve endings and the synthesis and structural modification of proteins at nerve endings. Both the usefulness and the limitations of the simple biochemical approach should be apparent from this description.

IV. Studies of the Effects of Inhibitors of Protein and RNA Synthesis on Learning and Memory

In the past few years we have undertaken studies of the effects on learning and memory in mice of actinomycin D, an inhibitor of cerebral RNA synthesis and of puromycin, cycloheximide, and acetoxycycloheximide inhibitors of cerebral protein synthesis. The studies with actinomycin D (Barondes & Jarvik, 1964; Cohen & Barondes, 1966) suggest that short term memory is not dependent on the synthesis of RNA molecules, but these conclusions are limited by the fact that the drug is highly toxic and that completely satisfactory experiments are not possible. Studies with puromycin (Barondes & Cohen, 1966, 1967a; Cohen, Ervin & Barondes, 1966; Cohen & Barondes, 1967) have been complicated by the finding that one effect of this drug on the brain is the production of occult seizures, which may interfere with memory storage. I therefore will discuss only our studies with acetoxycycloheximide (Barondes & Cohen, 1967b). This drug is an extremely potent inhibitor of cerebral protein synthesis. Five hours after intracerebral injection of a total of 20 μg of acetoxycycloheximide into the brain of mice, approximately 95% of the whole cerebral protein synthesis is inhibited, and inhibition at approximately this level is maintained for a number of hours thereafter. We have trained mice, at a time when maximal inhibition of cerebral protein synthesis has been established, to choose the left limb of a simple T-maze to escape shock to a criterion of three out of four consecutive correct responses.

We found that mice injected with acetoxycycloheximide or with saline learned the task in approximately six trials and that their acquisition curves were superimposable. When tested for retention 3 hr later both the saline and the acetoxycycloheximide injected mice had identical savings. Six hours after injection, however, the acetoxycycloheximide-injected mice showed markedly impaired retention, which was observed thereafter. Indeed, when similar mice were tested 6 weeks after injection no evidence of recovery of memory was found in acetoxycycloheximide injected mice, whereas saline-injected mice still had about 75% savings. These experiments suggest that protein synthesis is not required for learning of this task or for short-term memory —that is, memory for at least 3 hr after training. However, the findings suggest the long-term memory storage, that is, storage for somewhat more than 3 hr after training, is dependent on the synthesis of new protein. These findings do not imply that long-term memory storage is first established between 3 and 6 hr after training. Rather they suggest that the acetoxycycloheximide treatment interferes with long-term memory storage but that the short-term process persists for at least 3 hr. It remains possible that the long-term process is established very shortly after training but that its absence in our studies is not apparent until the short-term process has terminated. In point of fact electroconvulsive shock has little effect on memory of this task if administered as early as 15 min after training, which suggests that the long-term process has been established within 15 min. One might conclude from this that the delayed amnesia that is observed is a measure of the duration of short-term memory of this task in mice.

We found that if mice were trained in the position habit to a criterion of nine out of ten consecutive correct responses rather than three out of four consecutive correct responses, no effect of acetoxycycloheximide could be demonstrated. We interpreted these findings to mean that the small amount of residual protein synthesis that was present in the acetoxycycloheximide animals might be sufficient to effect long-term memory storage when there was considerable overtraining. We recently conducted similar experiments using a light-dark discrimination rather than a position discrimination (Cohen and Barondes, 1968). We find, using this task, that mice injected with acetoxycycloheximide 5 hr before training learn normally, remember normally 3 hr after training, and have amnesia 6 hr after training and thereafter even when training has been conducted to a criterion of nine out of ten consecutive correct responses. Therefore, in this task (which is more difficult to learn) acetoxycycloheximide interferes with memory even after prolonged training, possibly because the small amount of residual protein synthesis is not sufficient to effect storage of the discrimination.

The experiments reported here suggest that protein synthesis is not necessary for learning or short-term memory but is required for long-term memory storage, but considerable additional experimentation is required to evaluate this tentative conclusion. The major point I would like to make is that by the use of a highly potent and highly specific agent we can learn a considerable amount about the temporal sequence of memory storage and about a possible mechanism for its mediation without an understanding of the details of neuronal circuitry involved or of the interesting behavioral complexities characteristic of learning and memory.

V. Studies of the Transport of Protein to Nerve Endings and of the Biosynthesis of Glycoproteins at Nerve Endings

One popular speculation I have stated is that memory storage is based on the synthesis or structural change of protein that facilitates synaptic connections. As I have mentioned, such a change might occur either at the presynaptic or the postsynaptic portion of the synapse. The postsynaptic portion seems to be ideally situated for changes based on protein synthesis since the microsomes, the major sites of protein synthesis, are found close to the postsynaptic membrane. In contrast, the nerve ending terminal of the presynaptic cell is devoid of microsomes and has been thought to be relatively inactive in the synthesis of protein. Instead the protein of the presynaptic end is believed to be synthesized in the cell body and then transported to the nerve terminal through the axoplasm. The delay necessitated by transport from the cell body to the nerve ending implies that protein synthesized in the presynaptic cell body and transported to the nerve ending might not arrive at the nerve ending rapidly enough to effect an alteration in the synapse that might mediate memory.

It seemed of interest to determine the rapidity with which the protein synthesized in cell bodies in the central nervous system of the mouse can arrive at their nerve endings. Since structural changes in proteins might be particularly important for short-term memory storage, we also were interested in possible structural changes in proteins at nerve endings. Because glycoproteins on cell surfaces might regulate intercellular relationships, we were particularly interested in the possibility that carbohydrates might be incorporated into proteins at nerve endings and thereby influence interneuronal relationships. We therefore injected either radioactive leucine, a precursor of protein, or radioactive glucosamine, precursor of glycoprotein, intracerebrally into mice and studied the incorporation of these materials into various subcellular fractions of mice including the soluble protein extracted from isolated nerve endings (Barondes, 1964, 1968a). We found that after intracerebral injection of radioactive leucine there was rapid incorporation into protein of all subcellular fractions of brain except nerve endings. Incorporation was maximal within 1-2 hr after injection in all the major subcellular fractions; the specific activity of these proteins declined progressively thereafter due to degradation. In contrast, the specific activity of the soluble protein of nerve endings was extremely low 1-2 hr after injection of radioactive leucine but rose progressively in the ensuing hours and days due to transport of protein through the axoplasm from the perikaryon. This process was studied in some detail in the first few hours after injection to see if some protein made its way to nerve endings within this relatively short period of time.

We found that within the first few hours after injection of radioactive leucine there indeed was some migration of protein to nerve endings (Barondes, 1968c), a result which suggests that there is relatively rapid communication of some protein from the perikaryon of some cells to their nerve endings. These cells may well be small neurons with short axons, and such neurons have been suggested as being of particular importance in memory storage by Young (1966).

The foregoing studies therefore show that there is some rapid transport of radioactive protein to nerve endings in the central nervous system of the mouse, and this rapidly transported protein might be important in mediating an alteration in synaptic function for long-term memory storage. After administration of radioactive glucosamine different results were found. In contrast with radioactive leucine, the radioactive glucosamine was promptly incorporated into protein at nerve endings (Barondes, 1968b). The radioactivity found in protein was associated both with glucosamine and sialic acid. This finding suggests that there is local incorporation of carbohydrates into protein at the nerve ending and also that a structural alteration in nerve-ending protein, by incorporation of carbohydrate, could promptly influence synaptic transmission at this site.

The studies of transport provide no evidence that protein synthesis is required for long-term memory storage or that incorporation of carbohydrate into protein mediates short-term memory storage. Rather they are presented as examples of the types of experiments stimulated by the speculations that I have discussed, which might provide important information about memory storage in the absence of an understanding of the overall phenomenological complexities of this process.

The conclusion I would like to draw from this discussion is that the speculations I have reviewed and the experiments that have been generated have produced some results worthy of consideration by those of us interested in memory. Considerable additional work is required before these speculations can be accepted seriously. The relatively crude biological approaches discussed here, however, do seem to offer some useful information in the study of the enormously complex and humbling phenomena of learning and memory.

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THE AMNESTIC SYNDROMES: Disturbances in Coding?

Karl H. Pribram

> The limits of my language mean
> the limits of my world.
>
> Wittgenstein, *Tractatus Logico-Philosophicus*, 1922

I. Introduction

I want to take this opportunity to dispel the myth that experimentally produced local brain lesions (especially ablations) do not affect memory functions, that is, learning and remembering. This conception, like so many in neuromythology, derives its strength from the fact that it is a half-truth. In this instance the idea rests largely on Lashley's (1929) contribution, *Brain Mechanisms and Intelligence*, and derives support from his later publication (1950), *In Search of the Engram*. Lashley presented evidence and made interpretations. I shall show here that his data have been superseded—thus the fanciful aspect of the current myth—but that his interpretations were extremely shrewd—thus the myth's persistence. To make the counterargument I will describe data from experiments made over the past 20 years. In my laboratories alone some 952 behaviorally tested rhesus monkeys have been subjected to selective brain operations during this period. These studies provide evidence that makes me think that the impairments in memory functions produced by local experimental lesions are best subsumed as deficiencies in input processing, and I will describe the evidence that demonstrates that memory traces become distributed widely within a sensory projection system. I will then argue that the mechanism of remembering critically involves input coding, both during storage and retrieval.

II. Classification of Lesion-Produced Memory Disturbances

As noted earlier, the experimental analysis of subhuman primate psychosurgical preparations has, contrary to popular opinion, uncovered a host of memory disturbances. The initial technique by which these brain-behavior relationships were established is called the method of the "intersect of sums" (Pribram, 1954), an extension of what Teuber named the method of "double dissociation" of signs of brain trauma. The intersect of sums method depends on classifying the behavioral deficit produced by cortical ablations into *yes* and *no* instances on the basis of some arbitrarily chosen criterion; then plotting on a brain map the total extent of tissue associated with each of the categories *ablated:deficit* and *not ablated:no deficit*; and finally finding the intersect of those two areas (essentially subtracting the *noes* from the *yeses-plus-noes*). This procedure is repeated for each type of behavior under quantitative consideration. The resulting map of localization of disturbances is then validated by making lesions restricted to the site determined by the intersect method and showing that the maximal behavioral deficit is obtained by the restricted lesion. (See Table I and Fig. 1.)

Once the neurobehavioral correlation has been established by the intersect of sums technique, two additional experimental steps are undertaken. First, holding the lesion constant, a series of variations is made of the task on which performance was found defective. These experimental manipulations determine the limits over which the brain-behavior disturbance correlations hold and thus allow reasonable constructions of models of the psychological processes impaired by the various surgical procedures.

Second, neuroanatomical and electrophysiological techniques are engaged to work out the relationships between the brain areas under examination and the rest of the nervous system. These experimental procedures allow the construction of reasonable models of the functions of the areas and of the mechanisms of impairment.

Table I

Simultaneous Visual Choice Reaction[a]

Operates without deficit			Operates with deficit			Nonoperate controls		
	Pre	Post		Pre	Post		Pre	Post
OP 1	200	0	PTO 1	120	272	C 1	790	80
OP 2	220	0	PTO 2	325	F	C 2	230	20
OP 3	380	0	PTO 3	180	F	C 3	750	20
LT 1	390	190	PTO 4	120	450	C 4	440	0
LT 2	300	150	T 1	940	F			
H 1	210	220	T 2	330	F			
HA	350	240	VTH 1	320	F			
FT 1	580	50	VTH 2	370	F			
FT 3	50	0	VTH 3	280	F			
FT 4	205	0	VTH 4	440	F			
FT 5	300	200	VT 1	240	F			
FT 6	250	100	VT 2	200	F			
DL 1	160	140	VT 3	200	890			
DL 2	540	150	VT 4	410	F			
DL 3	300	240	VT 5	210	F			
DL 4	120	100						
MV 1	110	0						
MV 2	150	10						
MV 3	290	130						
MV 4	230	10						
MV 5	280	120						
CIN 1	120	80						
CIN 2	400	60						
CIN 3	115	74						
CIN 4	240	140						

[a] Pre- and postoperative scores on a simultaneous visual choice reaction of the animals whose brains are diagrammed in Fig. 2, indicating the number of trials taken to reach a criterion of 90% correct on 100 consecutive trials. Deficit is defined as a larger number of trials taken in the "retention" test than in original learning. (The misplacement of the score H 1 does not change the overall results as given in the text.)

Two major classes of memory disturbance have been delineated by these operations: *specific* and *contextual* amnesias.

III. The Specific Amnesias

Between the sensory projection areas of the primate cerebral mantle lies a vast expanse of parieto-temporo-preoccipital cortex. Clinical observation has assigned disturbance of many gnostic and language functions to lesions of this expanse. Experimental psychosurgical

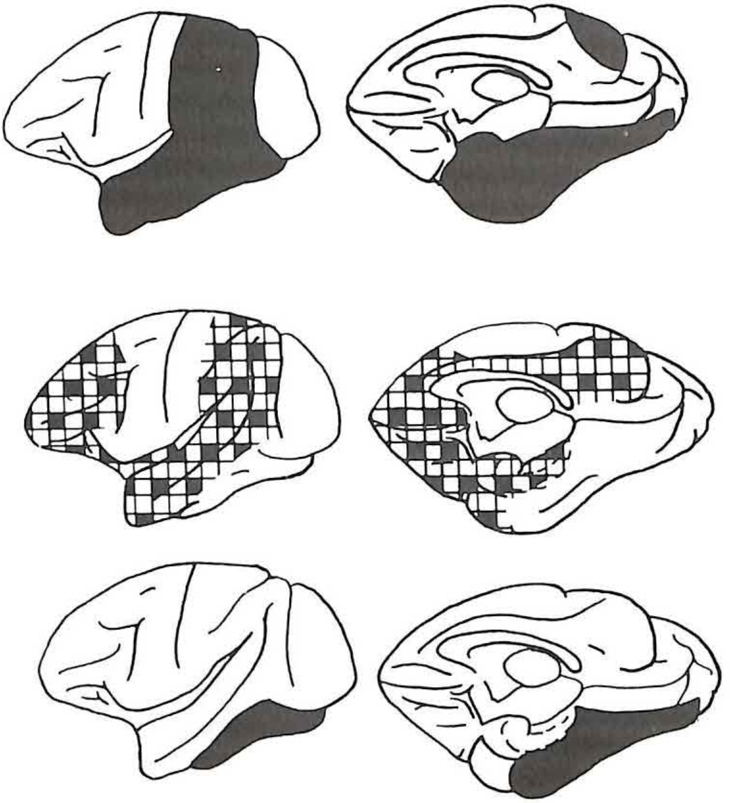

Fig. 1. The upper diagram represents the sum of the areas of resection of all of the animals grouped as showing deficit. The middle diagram represents the sum of the areas of resection of all of the animals grouped as showing no deficit. The lower diagram represents the intersect of the area shown in black in the upper diagram and that *not* checkerboarded in the middle diagram. This intersect represents the area invariably implicated in visual choice behavior in these experiments.

analysis in subhuman primates of course, is limited to nonverbal behavior; within this limitation, however, a set of sensory-specific agnosias (discrimination disabilities and losses in the capacity to identify cues) have been produced. Distinct regions of primate cortex have been shown to be involved in each of the modality-specific mnemonic functions: anterior temporal in gustation (Bagshaw & Pribram, 1953), inferior temporal in vision (Mishkin & Pribram, 1954), midtemporal in audition (Weiskrantz & Mishkin, 1958; Dewson, Pribram & Lynch, in press), and occipitoparietal in somesthesis (Pribram & Barry, 1956). In each instance discriminations learned prior to surgical interference are lost to the subject postoperatively and great difficulty (using a "savings" criterion) in reaquisition is experienced, if task solution is possible at all.

The behavioral analysis of these "specific" amnesias is still underway, but an outline of the psychological process involved can be discussed. Perhaps the easiest way to communicate this outline is to detail the observations, thinking, and experiments that led to our present view of the function of the inferior temporal cortex in vision.

IV. Search and Sampling

All sorts of differences in the physical dimensions of the stimulus, for example, size (Fig. 2), are distinguished less after the lesion (Mishkin & Hall, 1955), but there is more to the disability than this—as illustrated in the following story:

One day while testing monkeys with such lesions at the Yerkes Laboratories at Orange Park, Florida, I sat down to rest from the chore of carrying a monkey the considerable distance between home cage and laboratory. The monkeys, including this one, were failing misera-

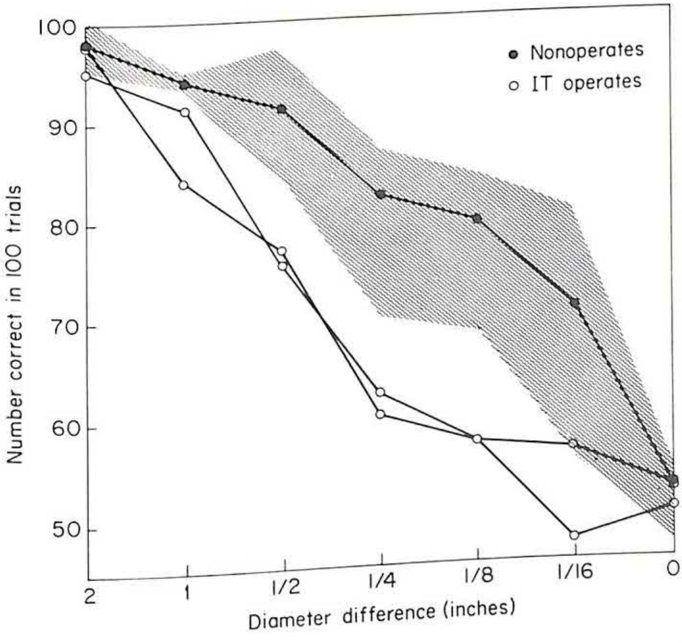

Fig. 2. Scores for two operates and four controls on the first run of size discrimination. Shaded area indicates the range of performance of the four nonoperate controls. IT operates = monkeys with resections of inferior temporal cortex.

bly at the visual discrimination tasks being administered. It was a hot, muggy, typical Florida summer afternoon and the air was swarming with gnats. My monkey reached out and caught a gnat. Without thinking I also reached for a gnat—and missed. The monkey reached out again, caught a gnat, and put it in his mouth. I reached out—missed! Finally the paradox of the situation forced itself on me. I took the beast back to the testing room: He was as deficient in making visual choices as ever. But when no choice was involved, the monkey's visually guided behavior appeared to be intact. This gave rise to the following experiment (Fig. 3), which Ettlinger (1957) carried out. On the basis of this particular observation we made the hypothesis that choice was the crucial variable responsible for the deficient discrimination following inferotemporal lesions. As long as a monkey does not have to make a choice, his visual performance should remain intact.

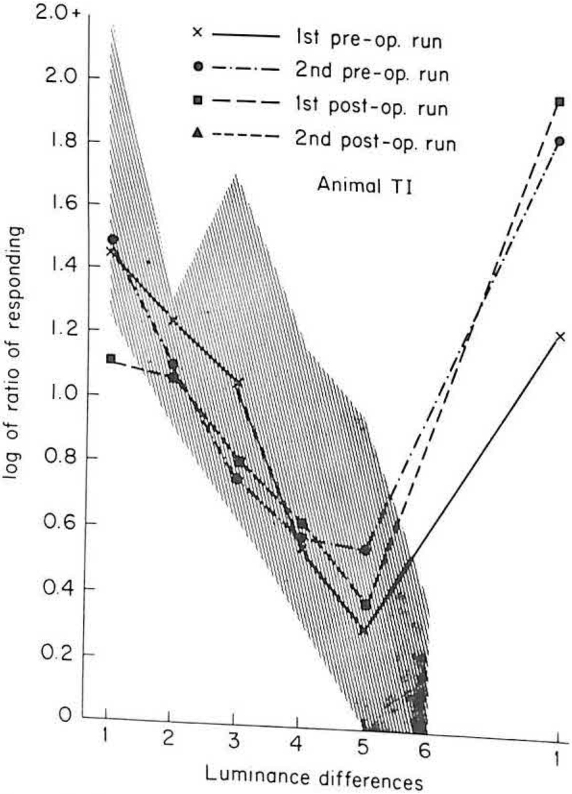

Fig. 3. Single manipulandum performance curves of a single animal in a varying brightness situation. Shaded area indicates variability among groups of four animals.

To test this hypothesis, monkeys were trained in a Gantzfeld made of a translucent light fixture large enough so the animal could be physically inserted into it. The animal could press a lever throughout the procedure but was rewarded only during the period when illumination was markedly increased for several seconds at a time. Soon response frequency became maximal during this "bright" period. Under such conditions no differences in performance were obtained between inferotemporally lesioned and control animals. The result tended to support the view that if an inferotemporally lesioned monkey did not have to make a choice he would show no deficit in behavior, since in another experiment (Mishkin & Pribram, 1954) the monkeys failed to respond differentially to differences in brightness.

In another instance (Pribram & Mishkin, 1955) we trained the monkeys on a very simple object discrimination test: an ashtray vs a tobacco tin (Fig. 4). These animals had been trained for 2 or 3 years before they were operated on and therefore were sophisticated problem-solvers; this, plus ease of task, accounts for the minimal deficit in the simultaneous choice task. (There are two types of successive discrimination: In one the animal has either to go or not to go, and in the other he has to go left or right.) When given the same cues successively the monkeys showed a deficit when compared with their controls, despite this demonstrated ability to differentiate the cues in the simultaneous situation.

This result further supported the idea that the problem for the operated monkeys was not so much in "seeing" but in usefully manipulating what they saw. Not only the stimulus conditions per se but the whole range of response determinants appear involved in specifying the deficit. To test this idea in a quantitative fashion we next asked whether the deficit would vary as a function of the *number* of alternatives in the situation (Pribram, 1959). The hope was that an informational measure of the deficit could be obtained. Actually something very different appeared when the number of errors was plotted against the number of alternatives (see Fig. 5).

If one plots repetitive errors made before the subject finds a peanut—that is, the number of times a monkey searches the same cue—vs the number of alternatives in the situation, one finds there is a hump in the curve, a stage where control subjects make many repetitive errors. The monkeys do learn the appropriate strategy, however, and go on to complete the task with facility. What intrigued me was that during this stage the monkeys with inferotemporal lesions were doing better than the controls! This seemed a paradox. As the test continued, however, after the controls no longer made so many errors, the le-

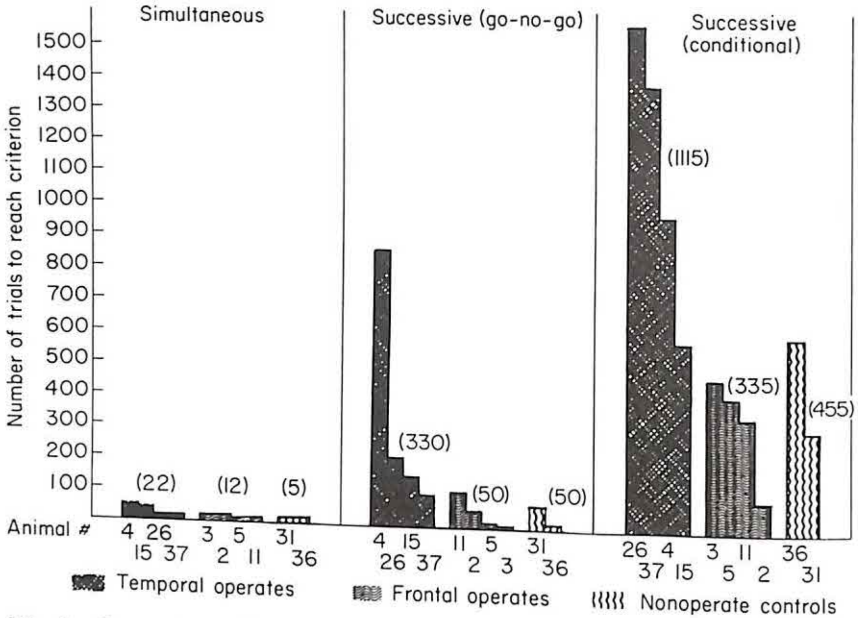

Fig. 4. Comparison of learning scores on three types of object discrimination by three groups of monkeys. Note that though the cues remain the same, changing the response which was demanded increased the deficit of the inferotemporal groups.

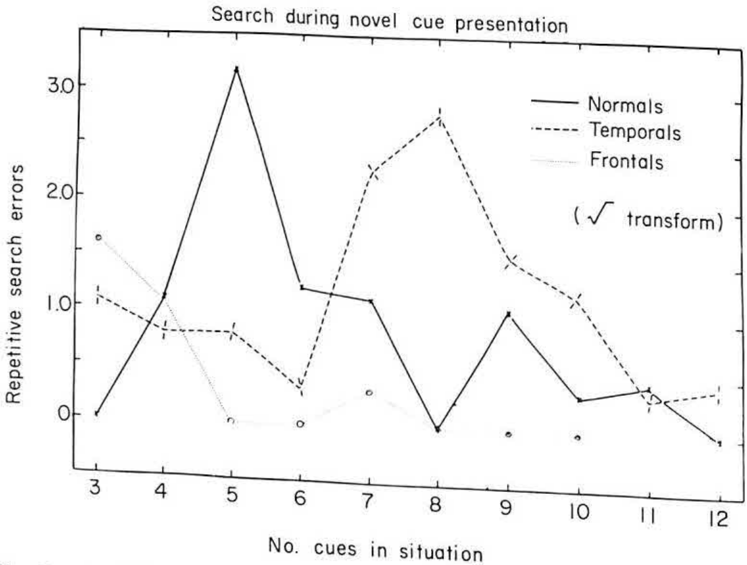

Fig. 5. Graph of the average number of repetitive errors made in the multiple object experiment during those search trials in each situation when the additional, that is, the novel, cue is first added.

sioned subjects began to accumulate an error hump even greater than that shown earlier by the controls.

When a stimulus sampling model was applied to the analysis of the data, a difference in sampling was found (Fig. 6). The monkeys with inferotemporal lesions showed a lowered sampling ratio; they sampled fewer cues during the first half of the experiment. Their defect can be characterized as a restriction in the visual field; however, the limitation is not in the visual-spatial field but in the information-processing field, that is, in the number of alternatives they can sample or handle at any one time.

In short, the modality-specific defect that results from a posterior "association" system lesion appears to produce an information-processing defect best described as a restriction on the number of alternatives searched and sampled.

V. The Contextual Amnesias

The second major division of the cerebral mantle to which mnestic functions have been assigned by clinical observation lies on the medial and basal surface of the brain and extends forward to include the poles of the frontal and temporal lobes. This frontolimbic portion of the hemisphere is cytoarchitecturally diverse. The expectation that different parts might be shown to subserve different functions therefore is even greater than that entertained for the apparently uniform posterior cortex. In the case of the posterior cortex the diversity of lesion effects nonetheless allowed classification: Differential discriminations were always involved, and the defects turned out to be sensory-mode specific. In the same manner, lesions of the frontolimbic region, irrespective of location (dorsolateral frontal, cingulate-medial frontal, orbitofrontal-caudate, temporal polar-amygdala, and hippocampal) have been shown to produce disruption of "delayed alternation" behavior. The alternation task demands that the subject alternate his responses between two cues (for example, between two places or between two objects) on successive trials. On any trial the correct response is dependent on the outcome of the previous response. This suggests that the critical variable which characterizes the task is its temporal organization. In turn, this leads to the supposition that the disruption of alternation behavior produced by frontolimbic lesions results from an impairment of the process by which the brain achieves its temporal organization. This supposition is in part con-

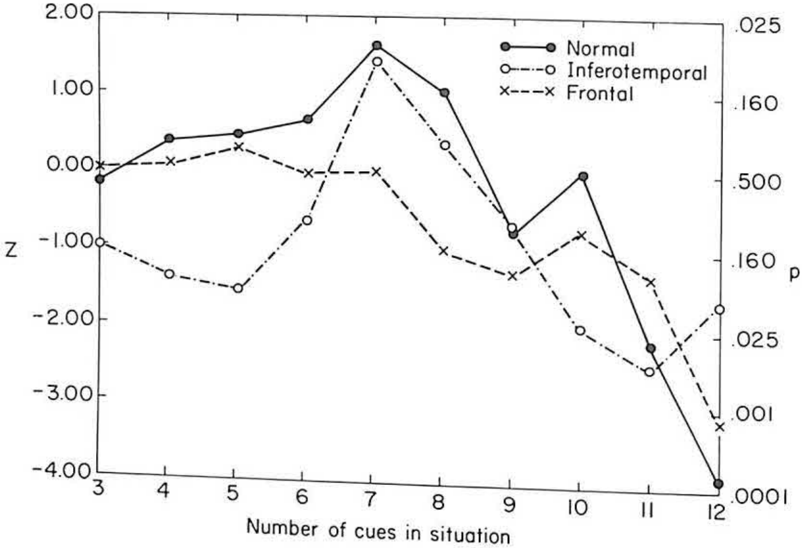

Fig. 6. Graph of the average proportion of objects (cues) that are sampled (except novel cue) by each of the groups in each of the situations. To sample, a monkey had to move an object until the content or lack of content of the food well was clearly visible to the experimenter. As was predicted, during the first half of the experiment the curve representing the sampling ratio of the posteriorly lesioned group differs significantly from the others at the 0.024 level (according to the nonparametric Mann-Whitney U Test).

firmed by further analysis, but severe restrictions on what is meant by "temporal organization" arise. For instance, skills are not affected by frontolimbic lesions, nor are discriminations of melodies. Retrieval of long-held memories also is little affected. Rather, shorter term mnestic processes are singularly involved. In animal experiments these are demonstrated especially clearly when tasks demand matching from memory a cue (as in the delayed response problem) or outcome (as in the alternation task) that in the past has shown some complexity in the regularity of its recurrence. Rather than identify an item, the organism must fit the present event into a "context" of prior occurrences, only some of which relate directly to the situation at hand.

As noted, different parts of the frontolimbic complex would, on the basis of their different structure, be expected to function somewhat differently within the category of short-term mnestic processes. Indeed, different forms of contextual amnesia are produced by different lesions. For instance, resection of the primate hippocampal formation (Douglas & Pribram, 1966) results in relative insensitivity to the nonreinforced aspects of the environment (the $S\Delta$ of operant conditioning; the negative instances of mathematical psychology). In their first experience with a discrimination learning situation these subjects show a peculiar retardation provided there are many nonrewarded alternatives in that situation: In a computer-controlled automated testing apparatus (DADTA) we face the subject with 16 panels; on only two of these are cues displayed, and pressing only one of them is rewarded. The site of display is randomized from trial to trial. Hippocampectomized monkeys press the unlit panels for thousands of trials after their unoperated controls have ceased responding to these "irrelevant" items. It is as if in the normal subject "ground" is established by progressively stronger "inattention" to those aspects of the situation that do not become "figures." This "inattention" is an active, evaluating process. For example, in a discrimination reversal task, when the demand is to notice the previously nonreinforced cue, unsophisticated subjects in our situation will press on almost any part of their cage and the testing apparatus before making an often-by-chance response to the nonrewarded cue.

I interpret these and many similar results to indicate that the hippocampal formation is involved in an evaluative mechanism by which "ground" or context is established.

VI. Expectancy, Novelty, and Registration

However, context formation, by itself, is of little use to an organism. Events must be fitted to context. A series of experiments on the orienting reaction to novelty and its habituation have pointed to the amygdala as an important locus in the "context-fitting" mechanism. Our experiments took off from those of Sokolov (1960).

Sokolov's experimental demonstration went as follows: Human subjects were exposed to a tone beep of a certain intensity and frequency, repeated at irregular intervals. Galvanic skin response (GSR), heart rate, finger and forehead plethysmograms, and electroencephalograms were recorded. Initially these records showed the perturbations that are classified together as the orienting response. After several repetitions of the tone, these perturbations diminish and finally vanish. This is habituation, a process that had been thought to reflect a *lowered* sensitivity of the central nervous system to inputs. But Sokolov now *decreased* the intensity of the tone beep, leaving the other parameters unchanged. Immediately the subjects again showed full-blown orienting responses. Sokolov reasoned that the central nervous system cannot be *less* sensitive—it is only less sensitive to *sameness*; to *differences* the central nervous system has become *more* sensitive. He tested this idea by rehabituating his subjects and then shortening the tone beep without changing any other parameter. As predicted, his subjects now oriented to the unexpected silence.

The orienting reaction and habituation are thus sensitive measures of an organism's expectancy process—the process by which context is organized. We therefore initiated a series of experiments to analyze in detail the neural mechanisms involved in orientation and habituation. This proved not as easy as it at first seemed. The dependent variables—behavior, GSR, plethysmogram, and electroencephalogram—are not as dependable as is desirable. As others have found, dissociation between them is readily observed when one makes one or another experimental variation. In our hands, forehead plethysmography turned out to be especially tricky. We finally settled on behavior, the GSR, heart and respiratory responses, and the electrical brain manifestations as most reliable.

The results of the first of these experiments (Schwartzbaum, Wilson, & Morrissette, 1961) indicate that, under certain conditions, removal of the amygdaloid complex can selectively affect the persistence of locomotor activity in monkeys by minimizing or retarding decrements that normally occur. The lesion thus produces a disturbance in the habituation of motor activity (Fig. 7).

The results of the experiments on the habituation of the GSR component of the orienting reaction (Bagshaw, Kimble, & Pribram, 1965) also indicate clearly that amygdalectomy has an effect (Fig. 8). The lesion interferes with the GSR in a situation in which the GSR is ordinarily obtained as part of the orienting reaction, although the GSR per se is not abolished. Concomitantly, deceleration of hearbeat, change in respiratory rhythm, and some aspects of the EEG indices of orienting also are found to be absent (Bagshaw & Benzies, 1968). From Schwartzbaum (1960a,b) and Bateson (in press) we know that the habituation of behavior is severely altered by these lesions. Putting the results together, one could conclude that the GSR component of orienting and heart and respiratory rate changes are in some way crucial to subsequent behavioral habituation. We therefore identified the process indicated by the autonomic components of the orienting reaction as "registering" the novel event.

However, the registration mechanism is not limited to novelty. Bagshaw (Bagshaw & Coppock, 1968) extended her work to a classical conditioning situation. Using the GSR as a measure of conditioning,

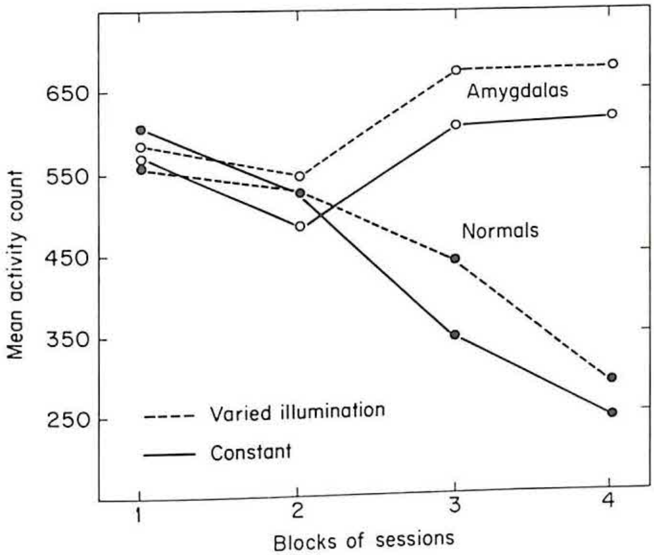

Fig. 7. Postoperative activity scores of normal and amygdalectomized monkeys for successive blocks of three sessions under conditions of constant illumination and more intense, varied illumination.

she found that normal monkeys not only condition but give more and more, as well as earlier and earlier, anticipatory GSR's in the situation. Amygdalectomized subjects fail to make such anticipatory responses. This observation suggests that registration entails some active process akin to rehearsal—some central mechanism aided by viscero-autonomic processes that maintains and distributes excitation over time.

Behavioral experiments support this suggestion. Amygdalectomized monkeys placed in the situations described above for testing the effects of resection of the hippocampal formation fail to take proper account of reinforced events. This deficiency is dramatically displayed whenever punishment, that is, negative reinforcement, is used. For instance, an early observation showed that baboons with such lesions will repeatedly—and this means day after day and week after week—put lighted matches in their mouths despite showing obvious signs of being burnt by them (Fulton, Pribram, Stevenson, & Wall, 1949). These observations were quantified in tasks measuring escape from and avoidance of shock (Pribram & Weiskrantz, 1957). The results of these two experiments have been confirmed in other laboratories and with other species so often that the idea has taken hold that loss of fear based on altered pain sensitivity is the primary difficulty that follows amygdalectomy. Bagshaw and Pribram (1968)

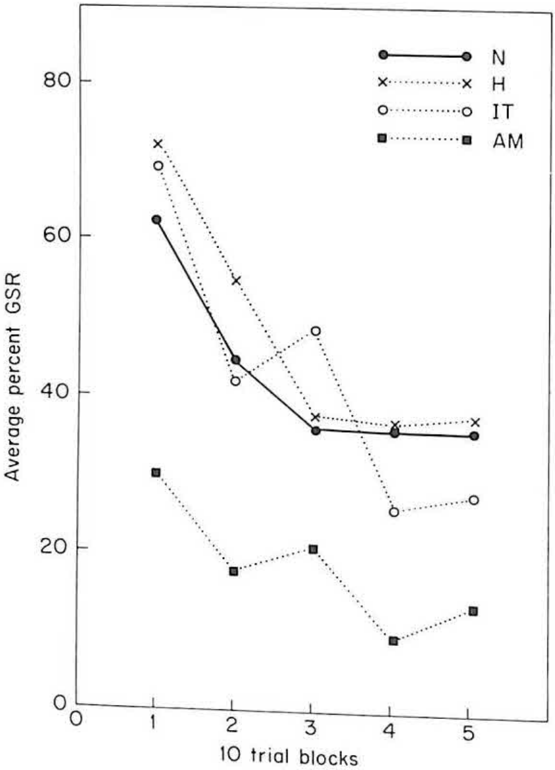

Fig. 8. Curves of percent GSR response to the first 50 presentations of the original stimulus for the normal (N) and three experimental groups (H, IT, AM), i.e., hippocampal, inferior temporal, and amygdala resected monkeys.

put this hypothesis to test and showed that the GSR threshold to shock, far from being elevated is, if anything, reduced by the ablation. This experimental result makes it possible to ascribe the observations that amygdalectomy produces a "loss of fear" directly to a mnestic disturbance—a disturbance of "registering" the noxious event and placing it in context—so that its recurrence will not on each occasion again be experienced as "novel."

VII. The Parsing Problem

The structures in the polar and medial part of the temporal lobe of primates are not the only ones concerned in short-term mnestic processes. Classically, disturbance of "immediate memory" has been ascribed to lesions of the frontal pole. Anterior and medial frontal resections were the first to be shown to produce impairment on delayed response and delayed alternation problems. In other tests of context-formation and fitting, frontal lesions also take their toll. Here also impairment of conditioned avoidance behavior and of classical conditioning and of the orienting GSR is found. Furthermore, error sensitivity was tested in an operant conditioning situation (Fig. 9). After several years of training on mixed and multiple schedules, 4 hrs of extinction were run, that is, the reinforcement (peanuts) was no longer delivered, although everything else in the situation remained the same. Note that the frontally lesioned animals failed to extinguish in the 4-hr period, whereas the control monkeys did (Pribram, 1961).

This failure in extinction accounts in part for poor performance in the alternation already described (Fig. 10): The frontally lesioned animals again make many more repetitive errors. Even though they do not find a peanut, they go right back and keep looking (Pribram, 1959).

This result was confirmed and amplified in a study by Wilson (1962). He analyzed the occasions for error – Did errors follow alterna-

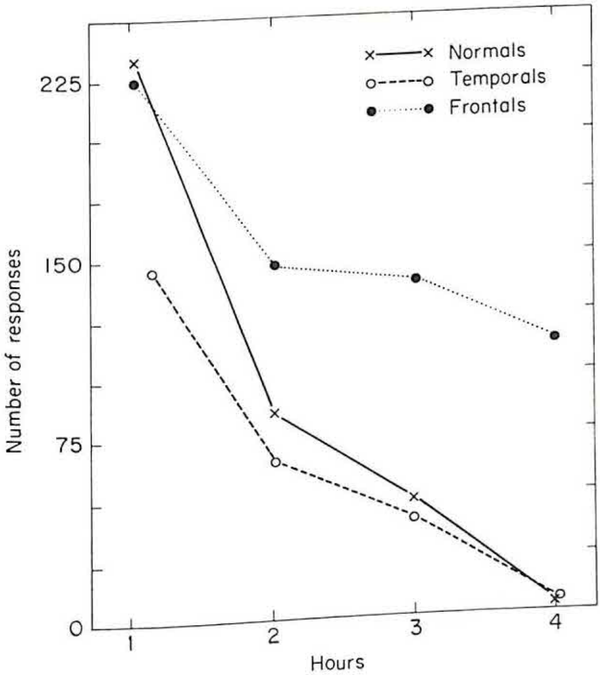

Fig. 9. Graph of performance of three groups of monkeys under conditions of extinction in a mixed schedule operant conditioning situation. Note the slower extinction of the frontally lesioned monkeys.

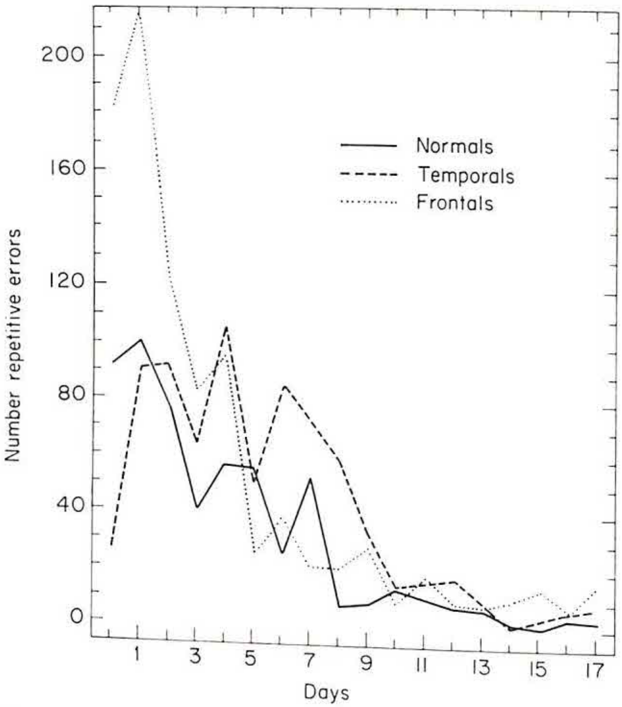

Fig. 10. Graph showing the differences in the number of repetitive errors made by groups of monkeys in a go, no-go type of delayed reaction experiment. Especially during the initial trials, frontally operated animals repeatedly return to the food well after exposure to the "nonrewarded" predelay cue. Note, however, that this variation of the delay problem is mastered easily by the frontally operated group.

tion or nonreinforcement? To determine which, he devised a situation in which both lids over the food well opened simultaneously, but the monkey could obtain the peanut only if he had opened the baited well. Thus the monkey was given "complete" information on every trial and the usual correction technique could be circumvented. With this apparatus the procedure was followed with four variations: correction-contingent, correction-noncontingent, noncorrection-contingent, and noncorrection-noncontingent. The contingency referred to is whether the position of the peanut depended on the prior correct or incorrect response of the monkey or whether its position was alternated independently of the monkey's behavior. Wilson then analyzed the relationship between an error and the trial preceding that error. Notice (Table II) that for the normal monkey the condition of reinforcement and nonreinforcement of the previous trial makes a differ-

Table II
Percentage of Alternation as a Function of Response and Outcome of Preceding Trial[a]

S	Preceding trial[b]			
	A-R	A-NR	NA-R	NA-NR
Normal				
394	53	56	40	45
396	54	53	36	49
398	49	69	27	48
384	61	83	33	72
Total	55	68	34	52
Frontal				
381	49	51	41	43
437	42	46	27	26
361	49	48	38	35
433	43	39	31	32
Total	46	46	33	33

[a] Comparison of the performance of frontally ablated and normal monkeys on alternations made subsequent to reinforced (R) and nonreinforced (NR) and an alternated (A) and nonalternated (NA) response.

[b] A, alternated; NA, did not alternate; R, was rewarded; and NR, was not rewarded.

ence, whereas for the frontally lesioned monkey this is not the case. Alternation affects both normal and frontal subjects about equally. In this situation, frontal subjects are simply uninfluenced by rewarding or nonrewarding consequences of their behavior.

Now let me return to the multiple choice experiment discussed earlier (Pribram, 1959). Here also this inefficacy of outcomes to influence behavior is demonstrated; it is illustrated (Fig. 11) by an increased number of trials to criterion after the monkeys have first found the peanut. The procedure calls for the strategy of return to the same object for five consecutive times, that is, to criterion. The frontally lesioned animals are markedly deficient in doing this. Again, we see that the conditions of reinforcement are relatively ineffective in shaping behavior once the frontal eugranular cortex has been removed, so that the monkeys' behavior is relatively random when compared to that of normal subjects (Pribram, Ahumada, Hartog, & Roos, 1964). Behavior of the frontally lesioned monkeys thus appears to be minimally controlled by its (repeatedly experienced and therefore expected) consequences.

In some respects, therefore, the frontal resection produces mnestic disturbances characteristic of both hippocampectomy and amygdalec-

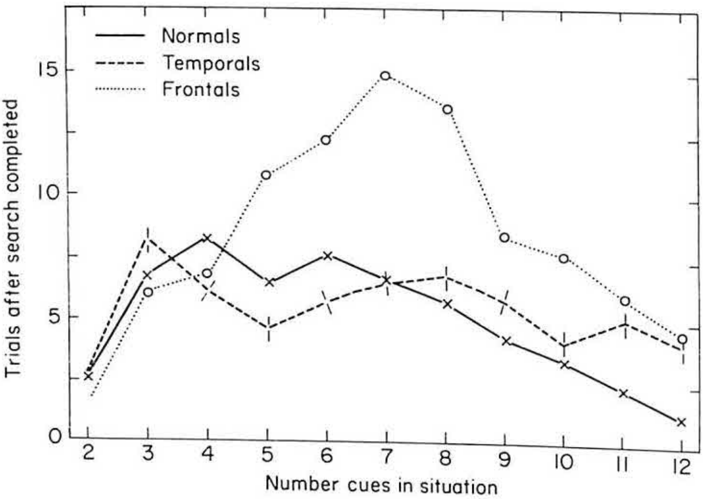

Fig. 11. Graph of the average number of trials to criterion taken in the multiple object experiment by each group in each of the situations after search was completed, that is, after the first correct response. Note the difference between the curves for the controls and for the frontally operated group, a difference that is significant at the .05 level by an analysis of variance ($F = 8.19$ for 2 and 6 *df*) according to McNemar's procedure performed on normalized (by square root transformation) raw scores.

tomy, though not so severe. Where temporal lobe ablations impair context formation and fitting, frontal lesions work their havoc on yet another contextual dimension. This is best demonstrated by manipulating the alternation task in a special way: Instead of interposing equal intervals between trials (R5″ L5″ R5″ L5″ R5″ L5″ . . .) as in the classic task, couplets of RL were formed by extending the intertrial interval to 15″ before each R trial (R5″ L15″ R5″ L15″ R5″ L15″ . . .). Immediately the performance of the frontally lesioned monkeys improved and was indistinguishable from that of their controls (Pribram & Tubbs, 1967). I interpret this result to mean that for the subject with a bilateral frontal ablation the alternation task becomes something like what this page would seem were there no spaces between words. The spaces, and the holes in doughnuts, provide some of the structure, the parcellation, parsing of events (doughnuts, alternations, and words) by which they become codable and decipherable.

VIII. How the Brain Controls Its Input

Recently much of our effort has been channeled into an attempt to clarify the neural mechanism disturbed by brain ablations. To this end a series of experiments was undertaken to find out how the brain cortex might affect the processing of visual information. It is appropriate to begin with some facts – or rather lack of facts – about the neuroanatomical relationships of the inferotemporal cortex. There is a dearth of neurological evidence linking this cortex to the known visual system, the geniculostriate system. There are no definitive anatomical inputs specific to the inferotemporal cortex from the visual cortex or the geniculate nucleus. Of course, connections can be traced via fibers that synapse twice in the preoccipital region, but connections also exist between the visual cortex, and the parietal lobe, the excision of which results in no change in visual behavior (as shown earlier). In addition, massive circumsection of the striate cortex does not impair visual discrimination (Chow, 1954; Pribram, Blehert, & Spinelli, 1966). Further evidence that these "corticocortical" connections are not the important ones can be seen from the following experiment. I performed (Table III) a crosshatch of the inferotemporal cortex much as Sperry (1959) had done earlier for the striate cortex and found no deficit either in visual learning or in performance. On the other hand, undercutting the inferotemporal cortex made a vast difference: It precluded both learning and performance of visual tasks. This suggests that the relationships of this cortex essential to visual behavior must come from somewhere below.

However, another proposal can be tested, viz., that the essential relations of the posterior association cortex are centrifugal, or efferent

Table III
Comparison of the Effects of Undercutting and Crosshatching Infero-Temporal Cortex of Monkeys on Their Performance in Several Discriminations

	Animal	3 vs 8	R vs G	3 vs 8
Crosshatch	158	380	82	0
	159	180	100	0
	161	580	50	0
	166	130	0	0
Undercut	163	[1014]	100	300
	164	[1030]	200	[500]
	167	704	50	0
	168	[1030]	150	[500]
Normal	160	280	100	0
	162	180	100	0
	165	280	100	0
	170	350	100	0

(Pribram, 1958). There is anatomical evidence to suggest and support such a notion. Some time ago two brains with inferotemporal resections were studied by Dr. Walle Nauta in his laboratory. These showed an efferent tract leading to the region of the superior colliculus, ending either within its substance or in the surrounding reticular formation (Nauta & Whitlock, 1954). No such fibers could be traced to the lateral geniculate nucleus. In support of this finding is a report by Kuypers (1962), who has also traced temporo-collicular fibers in monkey. The idea of an efferent mechanism "gating," or otherwise "partitioning," the input to the geniculostriate system has some backing as an explanation for the process of selective attention. How would an efferent mechanism of this sort work? To find out we performed the following experiment.

Instead of making ablations or implanting an epileptogenic lesion, we now chronically and continuously stimulate the brain. Dr. D. N. Spinelli in my laboratory designed the stimulator (Fig. 12) and the recording equipment (Spinelli & Pribram, 1966). The stimulator is sufficiently small so that it can be implanted under the scalp. It puts out a square-wave bidirectional pulse, 1 msec in duration and about 3 V in amplitude. The frequency of stimulation is approximately 8–10 pulses/sec. The batteries that drive the stimulator are rechargeable.

Records were made in the awake monkey (Fig. 13). Paired flashes are presented, and recordings are made from electrodes implanted in the occipital cortex. The response to 50 such paired flashes are accumulated on a computer for average transients. The flash–flash interval is varied from 25 to 200 msec. All are records from striate (visual) cortex. The top traces were recorded prior to the onset of stimulation and the lower ones after stimulation of the inferotemporal region had begun. Note that with cortical stimulation the recovery function is depressed, that is, recovery is delayed.

Figure 14 shows the average of such effects in five subjects. Chronic stimulation of the inferotemporal cortex produces a marked increase in the processing time taken by cells in the visual system.

A parallel experiment in the auditory system was done in collaboration with Dr. James Dewson. In this study, made with cats, removals of the auditory homologue of the inferotemporal cortex were performed. This homologue is the insular–temporal region of the cat. Dewson (1964) had shown that its removal impairs complex auditory discrimination (speech sounds), leaving simple auditory discriminations (pitch and loudness) intact. Removal, in addition, alters paired-click recovery cycles recorded as far peripherally as the cochlear nucleus. Bilateral ablation shortens the recovery cycle markedly. Of

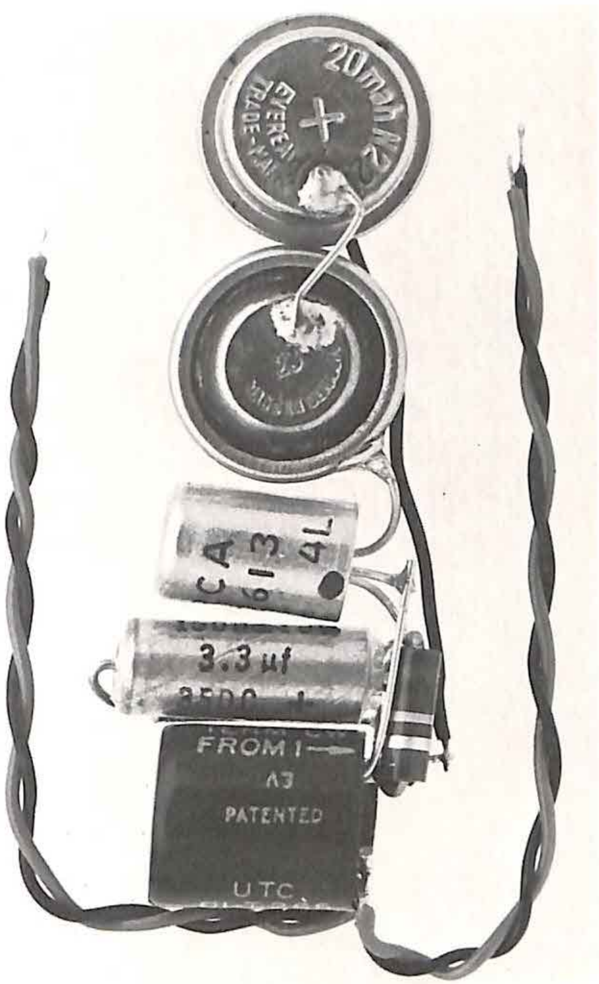

Fig. 12. Stimulator and batteries for chronic brain stimulation. Batteries are rechargeable nickel-cadmium and are available in different sizes from the manufacturer.

course, control ablations of the primary auditory projection cortex and elsewhere have no such effect. Thus we have evidence that chronic stimulation of the "association" cortex selectively prolongs, while ablation selectively shortens, the recovery time of cells in the related primary sensory projection system.

These results have been extended in both the auditory and visual modes. Electrode studies have shown alterations of visual receptive fields recorded from units at the optic nerve, geniculate and cortical levels of the visual projection systems produced by electrical

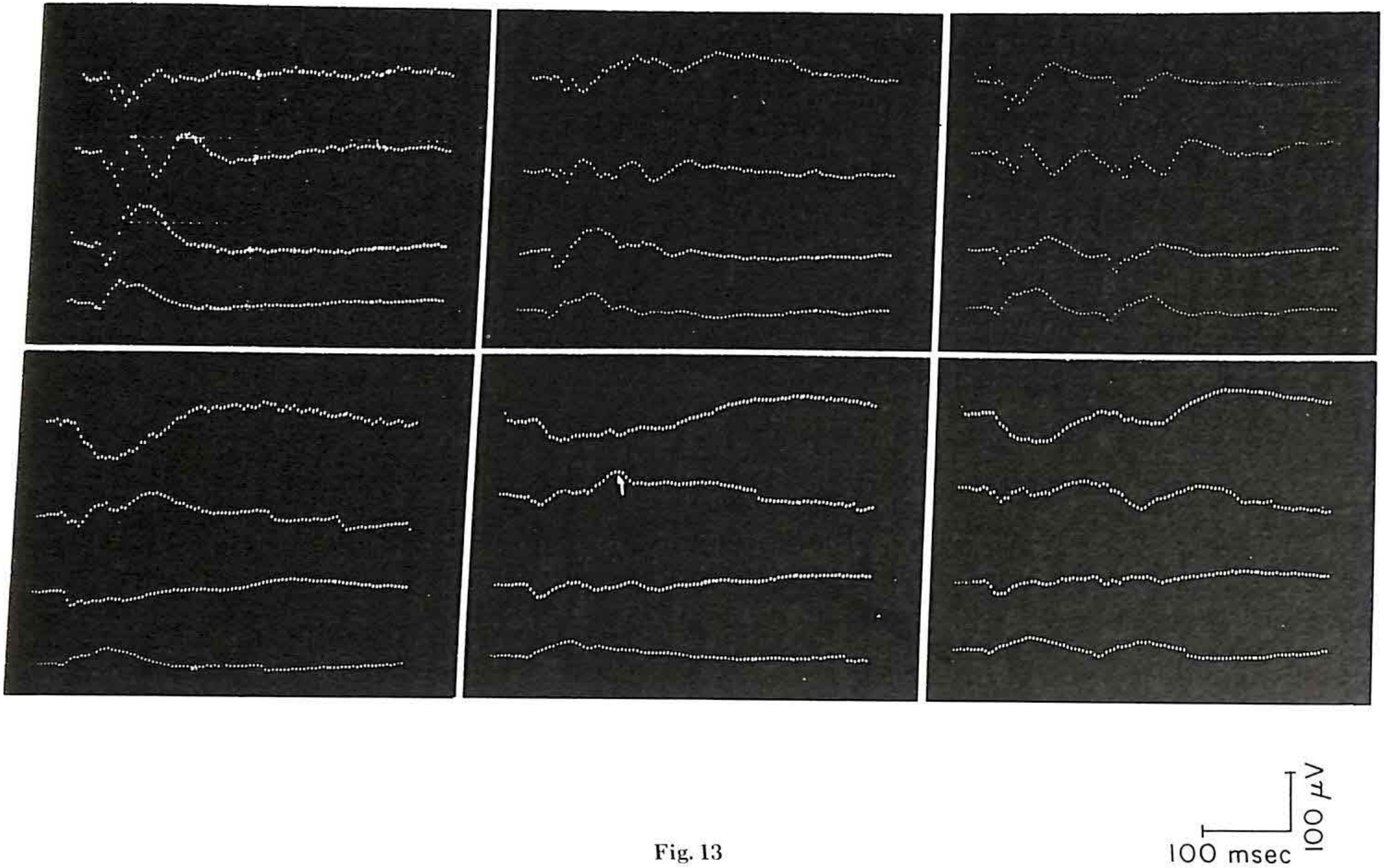

Fig. 13

stimulation of the inferior temporal cortex. The anatomy of the corticofugal pathways of these controls over sensory input also is under study. In the auditory system the fibers lead to the inferior colliculus and from there (in part via the superior olive) to the cochlear nucleus (Dewson, Noble, & Pribram, 1966). Definitive results as yet have not been achieved in our studies of the visual pathways, but preliminary indications lead to the pretectal-collicular region as the site of interaction between the corticofugal control mechanism and the visual input system.

The contextual amnesias only recently have become subject to neurophysiological analysis. Again, as in the case of the specific amnesias, cortico-fugal, efferent control mechanisms have been demonstrated. Results obtained in my laboratory show that in many instances these controls are the reciprocals of those involved in the sensory-mode specific processes (Spinelli & Pribram, 1967). Others (*Brain Res.*, 1967) have shown that the most likely pathways of operation of the frontolimbic mechanisms involve the brainstem reticular formation. Here, however, as in the case of the specific amnesias, control can be exerted as far peripherally as the primary sensory neuron (Spinelli & Pribram, 1967; Spinelli, Pribram, & Weingarten, 1965).

In general terms, the model derived from these experiments states that the operation of efferents from sensory-specific posterior systems tend to reduce, and those from the frontolimbic systems tend to enhance, redundancy in the input channels, that is, the primary projection systems. This presumably is accomplished by inhibition and disinhibition of the ongoing interneuronal regulatory processes within the afferent channels, both those by which neurons regulate the activities of their neighbors and those which decrease a neuron's own activity.

IX. The Distribution of Information in the Brain

This is not the first time in the history of experimental brain research that data have led investigators of complex mnestic disorders to

Fig. 13. A representative record of the change produced in visually evoked responses by chronic stimulation of the inferotemporal cortex. Upper set of records was taken before stimulation; lower set, during stimulation. All traces were recorded from the visual cortex; the first set in response to a single flash; the second, to flashes separated by 75 msec; and the third, to flashes separated by 150 msec. Actually this was the first of our series of experiments that called our attention to the changed recovery phenomenon. Note here also the change in wave form of the response even when a single flash was presented. However, this change did not appear in all of our subjects.

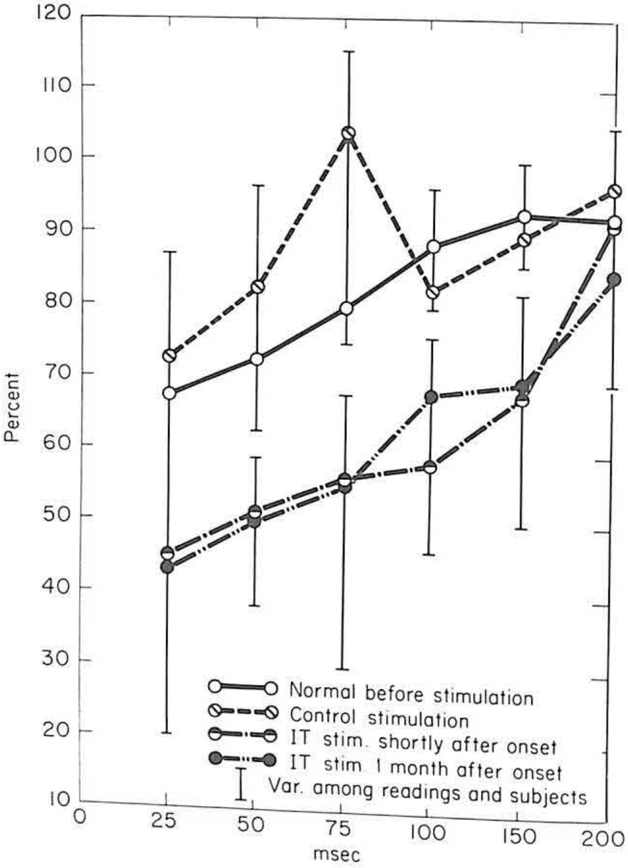

Fig. 14. A plot of the recovery functions obtained in five monkeys before and during chronic cortical stimulation: relative amplitude of the second response as a function of interflash interval.

focus on the primary projection systems. Munk (1881), von Monakov (1914), and Lashley (1942) pursued this course from an early emphasis on the "association" to a later recognition of the importance of the organization of the input systems. Of special interest in this pursuit are the experiments of Lashley that demonstrated that pattern vision remains intact after extensive resection—up to 85%—of the optic cortex. These results make it imperative to assume that input information becomes widely distributed within the visual system. Two types of mechanism have been proposed to account for such distribution (Pribram, 1969); here I want to present evidence that it indeed does occur.

We trained monkeys to discriminate between a circle and a set of vertical stripes by pressing the right or left half of a plastic panel upon which the cues were briefly projected (for 0.01 msec). Transient electrical responses meanwhile recorded from small wire electrical response were related by computer analysis to the stimulus, response, and reinforcement contingency of the experiment (Pribram, Spinelli, & Kamback, 1967). Thus we could distinguish from the record whether the monkey had looked at a circle or at the stripes, whether he had obtained a reward or made an error, and whether he was about to press the right or the left leaf of the panel. Interestingly enough, not all of these brain patterns were recorded from all of the electrode locations: From some, input-related patterns were obtained best; from others, the reinforcement-related patterns were derived; and still others gave us the patterns that were response-related. This was despite the fact that all placements were within the primary visual system, which is characterized anatomically by being homotopic with the retina. It appears therefore not only that optic events are distributed widely over the system but that response and reinforcement-related events reliably reach the input systems. Such results surely shake further one's confidence in the ordinary view that input events must be transmitted to the "association" areas for associative learning to be effected.

X. The Mechanism of Remembering

The experimental findings detailed here allow one to specify a possible mechanism to account for the lesion-produced amnesias. On the basis of the neurobehavioral and neuroanatomical data, I had suggested earlier (Pribram, 1960b) that the posterior "association" cortex by way of efferent tracts leading to the brainstem [most likely to the colliculi or surrounding reticular formation (Pribram, 1958)] partitions the events that occur in the sensory-specific system and classifies these events according to one or another scheme. During the course of our joint work, Dr. Spinelli would repeatedly ask: "What do you mean by 'partitioning'? What is 'partitioning' in neurological terms?" Until we had accomplished our electrophysiological experiments, I really had no idea just how to answer. But once we saw the results of these experiments, the neurophysiological explanation became evident: Partitioning must work something like a multiplexing circuit. In neurophysiological terms, when the recovery time of neurons in the sensory projection system is increased by posterior "association" cortex stimulation, fewer cells are available at any given moment to receive the concurrent input. Each of a successive series of inputs thus will find a different set of cells in the system available to excitation. There is a good deal of evidence that, in the visual system at least, there is plenty of reserve capacity—redundancy—so that information transmission is not, under ordinary circumstances, hampered by such "narrowing" of the channel (Attneave, 1954). Ordinarily a particular input excites a great number of fibers in the channel, ensuring replication of transmitted information. Just as lateral inhibition in the retina has the effect of reducing redundancy (Barlow, 1961), so the operation of the sensory-specific posterior "association" cortex increases the density of information within the input channel.

Conversely, the functions of the frontolimbic mechanism enhance redundancy, making more cells available at any given moment to concurrent input. This diminishes the density of information processed at any moment and enhances temporal resolution.

The model has several important implications: First, the nonrecovered cells, the ones that are still occupied by excitation initiated by prior inputs, will act as a context or short-term memory buffer, against which the current input is matched. A match–mismatch operation of this sort is demanded by models of the process of recognition and selective attention spelled out on other occasions by Craik (1943), Sokolov (1960), Bruner (1957), MacKay (1956), and myself (1960a, 1963a,b). These "occupied" cells thus form the matrix of "uncertainty" that shapes the pattern of potential information, that is, the "unexpectancy" that determines the selection of input signals that might or might not occur. The normal functions of the posterior cortex are assumed to increase the complexity of this context while those of the frontolimbic systems would simplify and thus allow readier registration and parceling.

Second, in a system of fixed size, reduction of redundancy increases the degree of correlation possible with the set of external inputs to the system, while enhancement of redundancy has the opposite effect. The number of alternatives or the complexity of the item to which an organism can attend is thereby controlled (Garner, 1962). This internal alteration in the functional structure of the classic sensory projection system thus allows attention to vary as a function of the spatial and temporal resolution that excitations can achieve, with the result that events of greater or lesser complexity can be attended to. The sharper the spatial resolution, the greater the "uncertainty" and, thus, the more likely that any set of inputs will be sampled for information. Conversely, the greater the temporal resolution, the more likely that attention is focused, and that events become grouped, memorable, and certain. In the extreme, the sharpening of the appetite for information becomes what the clinical neurologist calls stimulus-binding. Its opposite is agnosia, the inability to identify events because they fail to fit the oversimplified context of the moment.

Third, this corticofugal model of the functions of the so-called association systems relieves us of the problem of higher and higher order infinite regress—an association area "homunculus" who synthesizes and abstracts from inputs, only to pass on these abstractions to a still higher "homunculus," perhaps the one who makes decisions, etc. Former ways of looking at the input-output relationships of the brain invariably have come up against this problem (implicit or explicit) of "little men" inside "little men."

According to the model presented here, there is no need for this type of infinite regress. The important functions of perception, decision, etc. are going on within the primary sensory and motor projection systems. Other brain regions such as the posterior sensory-specific associated systems and the frontolimbic systems exert their effects by altering the functional organization of the primary systems. Thus these *associated* systems are not "association" systems; they simply alter the configuration of input-output relationships processed by the projection systems. In computer language the associated systems function by supplying *subroutines* in a hierarchy of programs, subroutines contained within and not superimposed from above on the more fundamental processes. In this fashion the infinite higher-order abstractive regress is avoided. One could argue that in its place a downward regress of sub- and subroutines is substituted. I would answer that this type of regress, through progressive differentiation, is the more understandable and manipulable of the two.

A final advantage of the model is that the signal itself is not altered; the invariant properties of a signal are unaffected (unless channel capacity is overreached). It is only the organization of the channel itself—the matrix within which the signal is transmitted—that is altered. Thus the same signal carries more or less information, depending on the "width" of the channel. I am here tempted to extrapolate and say that the signal carries different meanings, depending on the particular structure or organization of the redundancy of the channel.

Concretely, the associated cortex is conceived to program, or to structure, an input channel. This is tantamount to saying that the input must be coded by the operation of this cortex. In its more fundamental aspects, computer programming is in large part a coding operation:

The change from direct machine operation through assembler to one of the more manipulable computer languages involves a progression from the setting of binary switches to conceptualizing combinations of such switch settings in "octal" code and then assembling the numerical octals into alphabetized words and phrases and finally parceling and parsing of phrases into sentences, routines, and subroutines. In essence these progressive coding operations minimize interference among like events by identifying and registering unique structures among the configurations of occurrence and recurrence of the events.

The evidence presented here makes it not unlikely that one function of the posterior and frontolimbic formations of the forebrain is to code events occurring within the input systems. As already noted, the distribution of information (dismembering) implies an encoding process that can reduplicate events without recourse to widespread random neural connections. Regrouping the distributed events (remembering) also implies some sort of coding operation—one similar to that used in decoding binary switch settings into an octal format.

An impaired coding process therefore would be expected to produce grave memory disturbances. The question is thus raised whether lesion-produced amnesias, specific and contextual, reflect primarily malfunctions of mechanism of coding and not the destruction of localized engrams.

XI. Summary

Experimentally produced local brain damage does demonstrably impair memory functions. However, the impairment apparently is not so much a removal of localized engrams as an interference with the mechanisms that code neural events so as to allow facile storage and retrieval. Thus, the evidence shows that anatomically the memory trace is distributed within a neural system by means of an encoding process, while as a function of decoding the engram is reassembled, that is, remembered. Thus, what and whether something is remembered is in large part dependent on how it is, and that it is, adequately coded.

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III

SOME GENERAL PRINCIPLES OF REMEMBERING AND FORGETTING

NEUROPSYCHOLOGICAL MODELS OF MEMORY

O. L. Zangwill

When Dr. George Talland did me the honor of inviting me to take part in this symposium, I assumed that he would wish me to write about the amnesic syndrome in which, like himself, I have long been interested. In consequence, I was distinctly taken aback when he asked me to introduce the topic of neuropsychological models of memory. I am no model builder and must confess to a good deal of dissatisfaction with many of the models proposed by others. Either they limit themselves to very simple aspects of memory, as in most models proposed by biologists (for example, Young, 1966), or they base themselves on communications theory (for example, Broadbent, 1958), which may be sensible up to a point but very difficult to reconcile with neurophysiology.[1] In either event we seem oddly remote from issues of the kind that those of us with an interest in amnesic states meet with in our clinical studies.

However, I would like to call attention to a model recently proposed by my colleague, Dr. Larry Weiskrantz, which does take account of the disorders of memory that can be induced experimentally in animals or observed clinically in man (Weiskrantz, 1966). This model is admittedly tentative but does seem to me to make a start in drawing together the findings of experimental and clinical study. At bottom, the assumptions upon which it is based are quite traditional: First there is the idea of memory traces, that is, physiological simulacra of past events; second, the distinction between short-term and long-term memory storage, with the correlated assumption that short-term and long-term storage depend on different physiological mechanisms (Hebb, 1949); and third, the idea that long-term memory traces undergo progressive strengthening or consolidation with passage of time. This latter idea, which goes back at least to Müller and Pilzecker (1900), has recently been discussed at some length by Deutsch (1962). Although not everyone accepts these assumptions, at least they have been accorded wide currency in contemporary psychological discussion.

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[1] Uttley (1965), it is true, has made a valiant effort to reconcile bits with brains, but his biophysics is unfortunately beyond me.

Weiskrantz's ideas about the relations between short-term storage, long-term storage, and the effects on memory of cerebral dysfunction produced by adverse conditions (for example, electroshock or head injury) are embodied in a schematic diagram (Fig. 1). This shows a sequence of short-term traces (STT), subject to rapid decay, together with the long-term traces (LTT) based upon them, subject to progressive consolidation with passage of time. In Weiskrantz's view, the STT must persist for a certain minimum period (of the order of 0.5 sec) in order for the corresponding LTT to be based upon it. This accords with clinical experience of the duration of permanent retrograde amnesia after head injury (Russell, 1959). The effects of adverse condi-

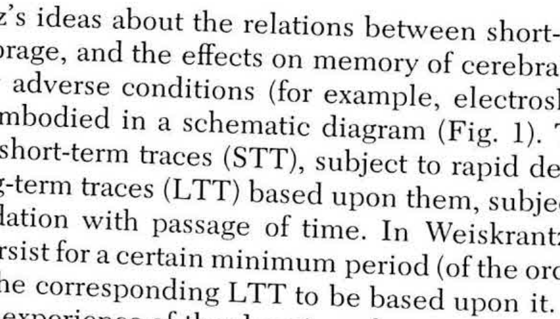

Fig. 1. Schematic diagram showing relationships among STT, LTT, and noise. It is assumed (see text) that LTT requires a short period of priming by STT before it can become viable. Arrows below abscissa indicate treatments of three different degrees of severity, A, B, C, giving rise to three different increases in noise level, with corresponding differences in degrees of retrograde and anterograde amnesia.

tions on memory are envisaged by Weiskrantz in terms of nonspecific interference with the retrieval mechanism and figuratively described in terms of "noise level." Three different "noise levels" are represented in the diagram, corresponding to arbitrary differences in the severity of the cerebral dysfunction. It will be seen that, as the "noise level" increases so does duration of amnesia in both its anterograde and retrograde aspects. It also will be seen that, in recovery from amnesia, memories will return in the reverse order of their establishment.

In arriving at this schema of memory, Weiskrantz has clearly been impressed by a number of observations, both his own and those of others. First there is the well-known dissociation between preservation of immediate memory and abolition of long-term memory in the amnesic syndrome (Talland, 1965; Milner, 1966). This undoubtedly suggests a "two-stage" process in the formation of the memory record. Second there is the striking observation by Bickford, Mulder, Dodge, Svien, and Rome (1958) that a degree of retrograde amnesia may be produced, in some cases at least, by electrical stimulation of the temporal lobe and that in these cases the duration of the amnesia is a function of the intensity of stimulation. This would seem to support Weiskrantz's view that the duration of retrograde amnesia is a function of the intensity of the disruptive agency. Third, there is the well-known "shrinkage" of retrograde amnesia that occurs in recovery from the acute effects of head injury and is commonly said to occur in order of time, the more remote memories returning first (Russell, 1959). This could be readily explained in terms of the postulated increase in strength of long-term traces with passage of time (consolidation) on the one hand, and the gradual decline of "noise level" on the other. Although the model in its present form is hardly precise enough to furnish an "explanation" of these phenomena, at least it would appear satisfactorily to embody them.

On the other hand, there are several features of amnesic states which would, I think, give difficulty to a model conceived along these lines. First, Weiskrantz appears to regard anterograde amnesia as due, primarily at least, to a failure of retrieval rather than to one of registration or storage. While there can be little doubt that retrograde amnesia (except for the very brief residual traumatic amnesias) reflects a retrieval failure, it is difficult to believe that the same can be true of anterograde amnesia. Were this so, much more should be recalled of recent events after remission, say, of a period of posttraumatic amnesia than is in fact the case. Second, it might be thought that after remission of a relatively prolonged amnesic state, for example, Korsakoff's syndrome, some events dating from the period of the illness should be available in memory after recovery. This has sometimes been claimed (Meggendorfer, 1928) but would appear very rare. Third, the model predicts that any condition giving rise to anterograde amnesia will at the same time produce retrograde amnesia and that the duration of both are related to the severity of the brain disorder. Whereas this in general is true (Russell, 1959), it should be borne in mind that quite extensive retrograde amnesias may persist after good recovery of memory for current events, as is sometimes seen after head injuries (Williams & Zangwill, 1952), tuberculous meningitis (Williams & Smith, 1954), or Wernicke's encephalopathy (personal observations). Such persisting retrograde amnesia can hardly be ascribed to residual general cerebral impairment, as is implied by Weiskrantz's concept of increased "noise level." It surely must be due to a retrieval failure in some part of the long-term storage system itself.

I also feel that Weiskrantz's model postulates too rigid a relationship between the "age" of a memory trace and its vulnerability to the effects of cerebral insult. The idea that ease of retrieval of a memory varies inversely with its recency was first developed by Ribot (1885) to account for the retrogressive nature of amnesia in progressive brain disease. While his so-called Law of Regression is broadly true, it should be borne in mind that most prolonged retrograde amnesias have a somewhat patchy character; "islands of memory" occur in an otherwise dense amnesia. Again, in studies of the restitution of memory after head injuries, Dr. Moyra Williams and I have shown that there is a good deal of irregularity in the return of memories, those which occurred shortly before the accident being by no means always the last to return. Indeed a patient might recall something that had happened a few minutes before the accident at a time at which his memory for the preceding hours, days or even weeks, was grossly defective. Not uncommonly, memory of recent events returned in a quite haphazard way, and these were gradually reconnected in orderly time sequence (Zangwill, 1964). Even when the retrograde amnesia had shrunk to its final, brief duration, appreciable retrieval defect for more recent pretraumatic events might remain in evidence (Williams & Zangwill, 1952).

Finally, as a former student of Sir Frederic Bartlett's, I must protest against Weiskrantz's apparent adherence to traditional "trace theory." I find it impossible to believe, as his model appears to suggest, that the long-term trace differs from the short-term trace only in its relative permanence. We know from the work of Bartlett (1932), Gomulicki (1956), and many others that long-term memory storage is highly selective and that long-term recall shows a strikingly abstractive character. Indeed there is good evidence that even comparatively short-term recall exhibits a considerable amount of abstraction (Gomułicki, 1956; Zangwill, 1956). How can a model of this kind explain these features? Oldfield (1954) has made one attempt—indeed the only one known to me—but his model is essentially based on communications theory. In any event, in trying to explain amnesia we evidently have to take account not only of the time relations of events in memory but also of the *form* in which the events themselves are stored.

Although I have been rather critical, I do not believe for a moment that models are unimportant or that empirical study can proceed without their construction and use. In spite of its sketchiness, Weiskrantz's model does embody important features of amnesia and consequently has genuine advantages to those whose approach to memory is from the clinical standpoint. I hope its author soon will revise it to overcome these difficulties and thereby come to generate a model with real predictive power.

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COMMUNICATION MODELS FOR MEMORY

D. E. Broadbent

Models for memory are of interest only insofar as they make a difference to the way we regard the problem, either by changing our techniques of observation or by giving us such an ability to predict that we give up observation altogether. The latter seems a dangerous effect at the present state of knowledge, so that one ought to tie any model closely to certain experimental methods. In summarizing rather dogmatically the model of memory that is supported by recent experiments of an information-processing kind, I therefore want to point particularly to the techniques used to assess memory in these experiments and to urge that the model can become relevant to pathology of memory only when those techniques are used in pathological cases. In general, this has not yet been the case, although I shall cite some relevant evidence about the failures of memory that occur as we get older.

The general model I would put forward, then, is one quite widely supported by different laboratories, although some of them might use other terminologies and differ on points of detail. It contains four stages of processing. The *first stage* is a memory store that is "preperceptual," that is, which contains all the information the sense organs provide about the world before it has been categorized or analyzed. This store receives the information directly from the senses, but it is not able to hold it for more than a second or so. After that time the information is lost forever, unless it has proceeded to the *second stage*.

This is a stage of coding or categorization, where one of a fixed set of alternative events occurs and the raw stimulus is thereby "pigeonholed." In concrete terms, if we show a man a dozen arabic numerals in a brief flash, he cannot correctly identify all of them, but he can pick whichever part of the visual field he is told to choose and identify the numeral at that point. He can do this with decreasing efficiency as we increase the time between the original flash and the instruction to report a particular part of the field. The stage of coding or identification is thus selective, not only in the sense that a numeral is identified as a "three" while its minor differences from other "threes" are ignored, but also in the sense that one part of the visual field is categorized and another is not.

The visual technique just mentioned has been explored by Sperling (1960) and others and obviously gives ways of attacking the efficiency of the first and second stages. An auditory technique that probably taps related mechanisms is one I have employed myself, in which one set of numbers is spoken to the right ear while a different set is spoken to the left. The listener can report quite readily all the numbers from one ear and then subsequently those from the other ear—and the latter apparently have been in some kind of buffer storage rather similar to the visual one of Sperling. Whereas performance on ordinary tests of digit span, such as remembering telephone numbers, remains reasonably constant as we get older, this type of memory for two simultaneous stimuli deteriorates sharply with age, as has been shown by Inglis and Caird (1963) and confirmed by Broadbent and Gregory (1965). At first sight this might suggest that age affects the first stage, the preperceptual store, but one must be cautious about this interpretation for three reasons.

First, that stage should be involved even in the orthodox memory span, which is much less affected by age. Second, evidence has been presented by Craik (1965) to show that some of the errors made in the Inglis situation are due to confusion of selection either in the coding stage or a later one of retrieval. Third, therefore, the bisensory situation does involve the coding stage as well as the first stage in such a way that the selective control of the information passing from first stage to second stage may be highly important, and it may be this control that deteriorates with age.

One line of evidence on this point comes from a condition used by Broadbent and Gregory (1965) in which three items were presented to the eye and three to the ear, but some of the items were digits and some were letters of the alphabet, and the two categories of item were to be recalled separately. Control of the set of coding categories employed thus was very important. This exaggerated the effect of age to the point where significant deterioration appeared between the age groups 16-25 and 25-35 years. To follow up this possibility would need the use of techniques that study directly the efficiency of the coding stage. Recently, for example, Broadbent (1967) analyzed data on the perception of words of different probability; misperceptions are themselves unequally likely to take the form of common rather than uncommon words, and from the pattern of errors and correct responses of various types one can show that the mechanism works in a way analogous to a statistical decision on noisy information. One can deduce also the biases that exist in the listener's coding stage. Such a technique, however, has not been applied as yet to pathological cases, even those due merely to aging.

Let us consider now what happens to information that has safely passed the hurdle of being encoded at the second of my four stages. It goes next into a *third stage,* which is again one of storage, but now of categorized and transformed information. At this stage the errors of memory which appear, even for visually presented material, may follow acoustic lines; one may see the letter B and remember the letter V presumably because one has said the letter to oneself (Conrad, 1964). For this reason, this stage is sometimes called the "echo box!" At this stage also memory becomes much less sensitive to the amount of time since the material arrived but is disrupted very seriously as any further events occur and are taken into the system (Waugh & Norman, 1965). The longer the information survives these hazards, the greater its chance of passing into a *fourth stage* where it is more permanently stored, but from which retrieval may be difficult. Such retrieval depends upon the establishment of a "tag" or label which, when later presented, will lead to the correct information among all the rest in the crowded fourth stage, whereas in the third stage the number of items present at any one time is bound to be limited. Finding information in this fourth stage may be especially hard if other information has previously or subsequently been stored under the same tag, but it is much less affected by the simple acoustic similarity that is important at the third stage (Baddeley, 1966).

In terms of a library model, the third stage is analogous to the "recent accessions" shelf, where one may find the last few books to arrive regardless of author or topic. The fourth stage corresponds to the main book stacks for which an index is required. To make the analogy with the human case more exact, one must suppose that some books may be dislodged from "recent accessions" by fresh arrivals and get lost without ever reaching the main stacks! The analogy implies that the third and fourth stages have different physical locations, but this is controversial. Some prefer to say merely that there are two different retrieval mechanisms from the same store.

What techniques are used to separate the third and fourth stages? One interesting method uses a relationship, developed by Murdock (1960, Conclusion XI) and by Tulving and Pearlstone (1966). If we present a man with a long series of words and ask him to recall them in his preferred order, he will when suitably trained remember a number of words that increase regularly with the number presented. That is,

$$\text{Number recalled} = A + B\,(\text{number presented})$$

(The relationship naturally only holds above the short presented lists for which number recalled is equal to number presented.)

In this relationship, the parameter A can be altered by some experimental conditions which do not affect B, and vice versa. For example, A is reduced by other activities intervening between presentation and recall; B is reduced by speeding up the presentation. The items contributing to A are those presented last and, in a subject who has learned to maximize his total score, are recalled first. We can regard A, therefore, as a measure of the third stage and B of the fourth. In work as yet unpublished by F. I. M. Craik, such an experiment has been performed with subjects of different ages, and B is reduced by aging rather than A.

This seems to put the effect of age very late in the chain of processes in our model: at the fourth stage, or perhaps most plausibly at the transfer from third to fourth (where some books had to be supposed to be lost from the library!). But once again we must beware of jumping to a conclusion: The problem of the fourth stage is one of retrieval, and Murdock's technique requires the memorizer to find for himself the words he is to recall. Recent work by Schonfield and Robertson (1966) has suggested that effects of age on memory are reduced if the testing technique is one of recognition rather than recall, so that retrieval is made much easier. This requires further investigation, but on present evidence the truth might well be that information passes safely to the fourth stage in old people but then cannot be retrieved. My own suspicion is that retrieval may employ the same selective encoding mechanism as does the second stage of intake of information; if so, a difficulty of retrieval with age would harmonize well with the suggestion that age impairs control of the second stage.

Two main morals seem to be in order. First, the time is not ripe for drawing detailed parallels between the components of an information-processing model and those of neural systems. It may seem tempting to point to the temporary and more permanent stores revealed by behavioral experiments and to equate them with structural changes in the neurons making permanent a temporary functional change. But long-term memory is not simply short-term memory crystallized into more durable form. Short-term memory is organized by simple sensory dimensions such as sound or time of arrival, long-term memory by meaning and association. The different patterns of error and facilitation in experiments on the two functions make this clear.

It is not at all obvious what physiological hypotheses could explain this change in character of older, established memories, although there is no lack of speculative mechanisms that would produce permanent memories organized in the same way as temporary ones.

Second, even a model of information processing as simple as this one requires several components to be operating in most memory tasks. Therefore, unless techniques are chosen with considerable care, a deterioration in performance may be wrongly interpreted as due to one component when it really is due to another.

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REPRESENTATIONAL SYSTEMS FOR STORING INFORMATION IN MEMORY[1]

Michael I. Posner

Abstract

A century of speculation in neurology, philosophy, and psychology has produced many ideas of how to describe representational systems for storing information in memory. These ideas are captured in such terms as memory trace, image, idea, schema, and motor program. Students of memory pathology have tended to use these words in describing the type of damage produced by some insult to the nervous system. Recent work in experimental psychology has begun to provide experimental operations in terms of which these representational systems can be defined. This paper seeks to outline a few of these techniques, some of the functional characteristics of the storage systems that the techniques reveal, and how these methods could be related to research into brain defects.

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The neural sciences have developed a variety of terms to describe the representation of information in memory. Such terms as memory trace, image, idea, schema, and motor program are by no means neutral. Each carries with it a theory of how some stimulus event comes to be represented in human memory. The theories may be as old and simple as the Platonic wax model that provides surplus meaning to the term "memory trace" or as new and complex as the digital computer that provides support for the notion of autonomous motor programs in the form of subroutines. This plethora of terminology and theory unfortunately has not been matched by a similar abundance of operations that can separate one form of memory code or representation from another. In the absence of such operations this rich language and theory has not been really accessible for the description of normal or pathological memory.

I would like to describe some experimental techniques that seek to provide an operational base for a number of rather different coding systems within human memory. The main goal is to suggest methods of experimentation that isolate various aspects of information storage. These are extensions of techniques that Welford (1958) and Talland (1965) already have used to explore the behavioral consequences of aging and Korsakoff's syndrome, respectively. As the occasion and my limited knowledge permit, I shall try to relate some of these experimental methods to the analysis of data obtained from clinical subjects.

In order to provide some organization to this paper I have tried in the first three sections to follow roughly the normal temporal sequence by which information presented to the organism is stored in memory. I want to emphasize that I do not believe that the sequence of stages suggested in this paper is fixed in the sense of being obligatory. Rather the subject (S) has considerable choice in the transformation of information from one to another coding system. Nonetheless, for the purposes of exposition it is useful to think first of a stage of information that is closely tied to the input modality, followed by an abstraction process requiring attention which, in the human, often takes the form of naming. When a series of items are abstracted in this way they are extremely likely to be lost unless there is available a system that can recycle them. Thus the first three sections concern unencoded information, naming, and rehearsal. These processes are relatively accessible both to experiment and introspection so long as the abstraction involves the use of simple names. However, when the abstracted information is in the form of motor programs, visual codes, or schema, the processes of experimentation and description become more difficult. In the last three sections I would like to deal with recent data on these three forms of memorial coding.

I. Unencoded Information

Most uses of the term *memory* refer to the storage of information that already has been perceived and analyzed at a fairly complex level. It is well known, however, that stimuli have a tendency to cause persistent neural activity in the sensory analyzer to which they are presented. Broadbent (1958) conceptualized this notion by suggesting that there is a peripheral storage system that preserves information when the *S*'s limited perceptual capacities are employed elsewhere. In particular, such a system could be used in cases in which the *S* was receiving simultaneous information to more than one sensory modality. The peripheral memory store preserves the information presented to one modality during the time in which *S* processes information presented to another channel.

A. VISUAL

Sperling (1960, 1963) and Mackworth (1963) have presented evidence for the persistence of a visual image during the first 1 or 2 sec after the offset of a visual stimulus. Sperling (1960) used large amounts of information and compared *S*'s performance when they were required to report all of it with the inferred storage capacity when a small part of it was sampled at random. Sperling (1963) and Mackworth (1963) studied the ability of *S*s to report letters after very short exposure times. Their results indicated the presence of a large-capacity, rapidly decaying store that could be erased by subsequent visual noise. The existence of a peripheral visual store generally has been accepted (Melton, 1963), perhaps because of strong evidence in the visual modality for the afterimage as a storage mechanism. There has been more controversy about the genuine character of peripheral storage of nonvisual information.

B. AUDITORY

Broadbent's original work on peripheral storage involved the auditory modality with information being presented simultaneously to the two ears. These studies indicated that under some circumstances one of the auditory channels could be delayed while *S* perceived and organized the other channel. Several types of recent experiments have provided additional evidence that such a mechanism is available in the auditory modality. For example, Ericksen and Johnson (1964) have shown that, upon receiving a cue, *S*s were able to search back in time to discover if a faint tone had occurred even though they had not previously perceived it. Experiments on recall of serial lists have shown that there are unique properties of the last few aurally presented stimuli. These unrehearsed items are relatively unaffected by presentation rate, repetition, and other organizational variables known to affect learning (Glanzer & Cunitz, 1966) but vary with stimulus intensity (Cohen, 1967) or delay prior to recall (Glanzer & Cunitz, 1966). These studies all suggest that aurally presented items also persist in a peripheral store until they can be processed centrally.

C. TACTILE

Recently Bliss, Crane, Mansfield, and Townsend (1966), using a method adopted from Sperling, provided some evidence for the peripheral storing of tactile information. He presented complex arrays of information in the form of blasts of air to the knuckles. He showed that, when *S*s were required to report only a part of the stimulus, their performance indicated that more information was available for brief periods of time after presentation than they could recall in a whole report.

D. SEPARATION OF STORES

In Broadbent's (1958) model it is suggested that the various sensory analyzers have available a common peripheral storage mechanism. If this is so, damage to that mechanism would be expected to produce a widespread inability to deal with temporal patterns from each analyzer. There is evidence that cortical damage may cause specific loss of the ability to respond to differential temporal patterns within a single modality (Neff, 1961). Moreover, it has been shown that visual noise will affect retention of prior visual information, but that auditory noise will not (Sperling, 1963). These findings appear to indicate that, at some level, peripheral storage systems are separate and that they may be thought of as being quite closely related to the individual sensory analyzers.

It is possible, however, that at another level there is a peripheral store (prior to perception) that involves a common neural code. It is difficult for psychological experiments to determine the identity or separability of peripheral stores since the individual characteristics of sensory systems make it hard to specify the expected properties of the common memory. For example, Bliss's analysis of the tactile system showed much less capacity than did Sperling's visual store. This could mean that the mechanisms are different, but it also could indicate differences in translation rate or spatial resolution that are properties of the particular tactile and visual memory codes used.

One approach is to compare the rate of decay of information in peripheral store that has been presented to different modalities. Most of these comparisons are indirect across different experiments using widely varying methods. It is common, however, for information to decay at very rapid rates (1–2 sec) regardless of modality (Broadbent, 1958; Sperling, 1963). Unfortunately, if decay rate is affected by information load and intensity, the comparison becomes quite tenuous. In one study (Ericksen & Johnson, 1964) using very small amounts of information and auditory presentation, some retention was found to 10 sec.

The most direct comparison between peripheral storage for different modalities has come from studies of simultaneous auditory and visual presentation of highly familiar material (Broadbent & Gregory, 1961; Margrain, 1967; Posner, 1967a). These studies have indicated that auditory items have greater persistence and show less loss from being delayed (held in peripheral store) than do the visual items. Recently Warren (1967) presented *S*s with mixed lists that included simultaneous auditory (dichotic), visual, and cross-modality pairs. He required *S*s to remember which digits were presented aurally and which visually. In this situation he found a greater overall error rate than with lists consisting entirely of audio-visual pairs. The overall advantage of the auditory items was maintained, but the proportion of cross-modality errors increased. This result may indicate that, at the level of peripheral storage being analyzed by these techniques, the mechanisms subserving the different modalities are not entirely separate. The question certainly merits further analysis with both normal and brain-damaged *S*s.

E. Coding

Murdock (1967) has reported that stimuli presented aurally show greater retention than visual items, even when there is adequate opportunity for *S*s to name all of the items upon presentation. The importance of the input modality in later recall emphasizes one point that has led to considerable confusion in the recent literature. The view that early stages of information processing prior to perception are closely associated with coding systems characteristic of a particular sensory modality should not be taken to mean that later stages of processing also might not be associated with these same sensory codes. In fact Broadbent (1958) indicated that material perceived and rehearsed still may be recirculated through a relatively peripheral mechanism. Sperling (1963), however, made an obligatory visual-to-auditory coding of information a feature of his model. While he intended to apply his analysis only to a restricted class of tasks, his results appeared to identify visual coding with peripheral storage in such a way as to make visual coding of stored information (imagery) unlikely. I wish to discuss this question in more detail in Section V.

It is certainly a frequent, if not a necessary, process for *S*s to take information that has been presented to a sensory modality and provide a verbal label for it. This process sometimes has been called read-in. In many cases we can identify it with the familiar act of *naming*.

II. Naming

It is a striking neurological deficit when a patient is able to perceive a familiar object but is unable to provide its name. In much of the experimental analysis of normal memory we think of the visual object and its name as being so closely linked as to be inseparable. Conrad (1964) illustrated the important role of the naming process in storing information when he showed that errors in written reproduction of visually presented letters could be predicted from an acoustic confusion matrix. Many studies of memory have shown that the type, rate, and familiarity of the verbal names attached to a stimulus are important determinants of its likelihood of recall. It is, however, unusual for psychologists interested in memory to study the naming process itself.

Despite its great familiarity and introspective ease, there can be no doubt that the process of naming a letter, word, or object is rather complex. About .3-.5 sec elapse between seeing a word or a picture of a familiar object and initiating a response to it. Producing the sound of the name consumes a roughly equivalent interval. Thus the process of naming even the simplest and most familiar object consumes more than half a second. This time allows for a large amount of processing and is sufficient to enable the operation of apparatus of great complexity. If techniques can be made available to isolate the components of the naming process, it may be possible to specify more precisely the behavioral deficit in certain forms of brain damage.

A. PROCESSING STAGES

In a recent experiment Sternberg (1967) presented *S*s with a single visual digit that was either clear or distorted by noise (a random checkerboard). He measured the speed with which they could tell whether this digit was or was not a member of a positive set consisting of one to four digits. The relationship between speed of classification and number of digits in the positive set was linear. The presence of noise affected both the intercept and slope of the function. Sternberg concluded that a visual pattern is first registered in a system that abstracts it by removing some or all of the noise. This stimulus is then matched against members of the positive array. Because noise can affect the time necessary for a match as well as registration, Sternberg concluded that a representation of the input can be matched with other materials prior to obtaining the digit name. Since the positive array in Sternberg's procedure is arbitrary, it is likely that his task proceeds much more slowly than normal naming,[2] but it does provide a technique for investigating stages of processing that are prior to determining the actual name.

In our laboratory we have developed some techniques that attack this question somewhat more directly (Posner & Mitchell, 1967). Our studies measure the reaction time (RT) taken for *S*s to classify a pair of letters as "same" or "different." At the simplest level of processing, a "same" response is based upon physical identity (for example, AA), at a higher level on name identity (for example, Aa), and at a still higher level of rule identity (for example, both vowels). What is particularly useful about this procedure is that the identical stimulus-response combination (for example, AB-"different") may be studied at all three levels of processing. As the depth of processing required for a "same" judgment increases, the time to respond "different" to AB changes from about 450 to 800 msec.

B. PHYSICAL IDENTITY

The physical identity match is closest to Sternberg's registration stage. It would be logically possible to obtain matches at this level even if S had never seen the stimuli before. The empirical results obtained with the matching technique have indicated its utility in allowing us to separate the various components involved in naming. For example, it has been shown that the time taken to name a picture is greatly affected by its familiarity (Oldfield & Wingfield, 1965) and that it also can be affected by brain damage (Oldfield, 1966). However, matching a pair of physically identical letters is not done faster than matching nonsense forms of similar complexity (Posner & Mitchell,

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[2] For studies of the search process with highly overlearned sets see Morin, Derosa, and Stultz (1967).

1967) or mirror image letters (Hochberg, 1968) despite vast differences in familiarity. Nor is it faster to match a pair of identical familiar pictures than a pair of identical unfamiliar pictures (Wingfield, 1966). Moreover, a letter appears to operate as a unit at the level of a physical match, since it is no faster to match single line slants (for example, / /) than it is to match letters (for example, AA) that consist of an aggregate of slants (Eichelman & Posner, 1967).[3] The physical identity match gets at the very early stage of the naming process. The lack of familiarity effects and of the requirement to search memory suggest that many types of brain damage that affect the naming process as a whole will not influence this component.

C. NAME IDENTITY

Matching two stimuli that are physically unrelated but have the same name (for example, Aa) requires that *S* use information stored in memory in order to match the figures. For letters it takes about 70-100 msec longer to match on the basis of name identity than on the basis of physical identity (Posner & Mitchell, 1967). In another experiment matching pairs of simultaneous visual digits and simultaneous auditory (dichotic) digits was compared with matching an audio-visual pair. The *S*s were instructed to respond "same" if the digits were identical. It took about 70 msec longer to deal with the audio-visual signal than with the average of the two same-modality pairs. Since these differences did not obtain when "same" was defined as both odd or both even, they did not appear to be due to switching between modalities but rather to the learned correspondence between a visual digit and its name as opposed to the physical identity of same modality pairs. Similarly, Wingfield (1966) has shown that the difference between matching two identical pictures and matching a picture with its name is also about 75 msec. Thus the time to go from the physical to the name level seems to have considerable generality with different materials. The time between physical matching and name matching includes two components. The first involves obtaining the names. This requires relating the present stimulus to stored information. Second is the process of matching the names. Brain damage could exert its influence on either or both of these components, but in most cases the first is more likely since it is of greater complexity. To accomplish the first component *S* must have the relevant information stored and must have the neural apparatus required to match the present input to the stored information. In order to separate these two functions one can investigate the ability of *S*s to perform analogue matches.

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[3] The unity of a single letter may break down when it is embedded in meaningful words (Eichelman & Posner, 1967).

D. Analogue Matches

It is possible to obtain matches that are slower than physical identity but faster than those based only upon the name. For example, the letter pair Cc can be matched much faster than Aa but not so fast as its physically identical controls (CC,cc). This appears to give us a means of investigating the processes of stretching, rotation, and so forth that must be performed upon the abstracted physical representation of the input in the process of arriving at the name (Sternberg, 1967). We have shown, for nonsense materials, that small differences between a stimulus pair such as variation in size or orientation will increase the time for them to be identified as "same." It would be of interest to know whether brain damage that affects naming letters also would affect these processes of matching similar but not identical pairs. If it did not it would suggest that the lesion had left intact the processes necessary to equate a new input with stored information concerning the physical form of the letter but had either obliterated or made unavailable the stored information itself.

E. Classification

If *S* is asked to say whether two letters are both vowels or both consonants, the time required to respond is uniformly longer than when he matches on the basis of name (Posner & Mitchell, 1967). Moreover, the introspective accounts given by the *S*s seem to indicate that they name the stimuli before making the classification. It is of considerable interest in understanding the sequencing of information processing to know the generality of this order. Are some classifications or associations made on the basis of the visual stimulus patterns and others based upon the name of the pattern? For example, can "J" be called a letter without first being named? Certain data from the neurological literature seem to indicate that naming is not a necessary stage in classification. For example, Geschwind (1965) argues that his patient might not be able to name a glass of water but would make other perfectly appropriate responses to it such as drinking from it when thirsty. However, this is a motor pattern rather than another verbal classification. Gazzaniga (1967) suggests that complex emotional patterns may be evoked in his split-brain patients without their being able to make any verbal account of the stimulus because there is no access to verbal storage. Perhaps the normal *S* can go directly from a visual pattern to complex associations without going through the name. The matching techniques I have outlined may allow us to answer this type of question.

F. Brain Damage

There exists a variety of hypotheses concerning information-processing deficits arising from various types of lesions. These include such notions as general slowing of activity (Birch, Belmont, & Karp, 1966), an inability to employ multiple sets (Talland, 1967), reduction in abstracting ability (Goldstein, 1948), loss of the ability to sequence behavior (apraxia; Gardner, 1958), "disconnection" between sensory modalities (Geschwind, 1965), and lack of access to verbal codes (Gazzaniga, 1967). Almost all of these hypotheses make predictions about which components of the naming and classification process ought to show impairment and under what conditions. The various matching tasks discussed in the last few pages provide tasks that tap at these components in a relatively pure way. It ought to be possible to use such tests as a means of detailing the specific components of the deficit a particular patient shows. Undoubtedly new techniques will continue to refine our knowledge of the naming process, and these will in turn provide more specific and detailed procedures for revealing the nature of cognitive deficits.

III. Attention and Rehearsal

Once a stimulus is coded into verbal form it may or may not survive in memory. The period during which a verbal stimulus is extremely liable to loss has been called short-term memory (Fitts & Posner, 1967). Much is known about the short-term retention of verbal items, and I will not review the literature here since many reviews are readily available (Atkinson & Shiffrin, 1968; Postman, 1964; Posner, 1963, 1967a).

A. Limited Capacity

An analysis of the role of attention in sustaining the verbal memory trace may tend to unify results obtained with humans with those obtained with other species and provide a deeper insight into the complex behavioral effects of brain damage. In many human studies rehearsal is held to be equivalent to silent speech. This view has three disadvantages. First, it places an impassable border between verbal and nonverbal organisms and between retention of material which is verbally coded and that which is not. Second, it means that rehearsal is thought of as all-or-none, that is, as either present or absent. Third, it requires reliance upon instructions or retrospective reports about whether a given condition allowed S to rehearse.

This notion can be replaced with a view of rehearsal as requiring a portion of some limited-capacity information-processing system available to all organisms. The idea of limited processing capacity not only has appeal on an introspective basis but has proved useful in uniting many strands of experimental and physiological research (Sanders, 1967). In the human being, the limited-capacity system may be defined by experiments on rate of information transfer (Posner, 1966a) or on the degree of interference between simultaneous tasks (Broadbent, 1958). Recent efforts have provided an increasingly quantitative account of the nature of man's limited capacity (Keele, 1967; Taylor, Lindsay, & Forbes, 1967).

It is clear that the retention of verbal items in short-term memory requires a portion of this limited-capacity system (Posner, 1966a). On this basis, therefore, they can be said to require rehearsal because any task that occupies a portion of *S*'s capacity will result in an increase in forgetting. It is more difficult to define such a system in infrahuman organisms, but recent analysis of delayed response and delayed matching to sample in the monkey seem to argue that the same type of relationship between attention during the interval and retention of prior material is present in that species (Drachman & Ommaya, 1964; Fletcher, 1965). Perhaps in the future such physiological indicants as the vascular changes accompanying orientation, pupil size changes, and EEG pattern will allow us better methods of establishing more detailed correspondences between central processing capacity in the human being and limited capacity mechanisms in other animals.

B. HIPPOCAMPAL DEFICIT

The close quantitative correspondence between retention and attention may help to shed some small amount of light upon the puzzling effects that occur in various organisms with hippocampal lesions. The human data have been interpreted as showing that the hippocampus is vitally concerned with the conversion of information from a short-term system that requires continuous processing to a longer term system that does not require that capacity (Drachman &

Arbit, 1966; Ojemann, 1964). This explanation seems somewhat plausible in the monkey, though in many situations there appears to be relatively little loss in the ability to learn new tasks. In the rat, however, several summaries of the recent literature seem to point to the role of the hippocampal lesion in reducing the ability of the rat to orient to new cues in the presence of previously rewarded signals from the environment (Douglas, 1967; Kimble, 1968; Douglas & Pribram, 1966). Moreover, some interpretations of data obtained by physiological recording from hippocampal cells also indicate that this structure is involved in the orienting response (Sokolov, 1966). Of course these findings have been obtained from widely different organisms and tasks, so that discrepancies in interpretation may reflect genuine physiological or functional differences. However, the close relationship between attention and retention in normal memory might well indicate that a general reduction in the capacity to process signals can result in deficits that appear sometimes as attentional and sometimes as memorial.

To illustrate what I mean let me try an anecdote that will be within the range of most of our introspections. The consumption of alcohol seems to have a generally depressing effect upon the human nervous system. While conversing in an alcoholic state it is my impression that I can either remember the conversation by virtue of great concentration upon it or can focus normal attention on ongoing events but have little recollection the following day of what was said. Studies of human (Carpenter & Ross, 1965) and animal (Van Laer, Jarvik, & Van Laer, 1965) performance after consumption of alcohol have been interpreted in terms of both attentional and memory deficits in much the same way as have studies of hippocampal lesions. I do not wish to suggest that the rat or the patient performs a conscious trade-off between attention and memory. However, it is possible that the organism and task determine the state of this trade-off and that the hippocampus is not exclusively associated with either function but instead contributes to the general capacity of the system. I would like to return to the hippocampus again in the next section in order to suggest some limitations on its involvement in memory.

It is important to point out that retention over short periods of time will not always require access to central processing capacity (rehearsal). For example, a single letter or a highly novel stimulus may be coded directly into long-term memory without undergoing any measurable period of rehearsal. Moreover, some types of material seem to be completely or largely unaffected by rehearsal. Indeed, I plan to discuss one such type in the next section.

IV. Motor Programs

There have been many suggestions that the mechanisms underlying the retention of motor skills might be rather different from those subserving verbal materials. The philosopher Gilbert Ryle (1949) made a distinction between "remembering that" and "remembering how" that reflects this notion. More recently experimental psychologists (Broadbent, 1958; Bruner, 1966) have speculated that storage systems for motor activity were different from those for verbal material or visual images. Broadbent (1958) suggested that the difference might lie in the reduced requirement of motor material for rehearsal by the central processing system.

In a series of studies (Posner & Konick, 1966; Posner, 1966a; Posner, 1967b) my colleagues and I attempted to provide an experimental analysis of these theoretical suggestions. I will review the results very briefly since more detailed analyses are readily available. In these studies we compared the retention of a visual point in space (visual location) or a visually guided motor movement (visual distance) with retention of a blind motor movement for which only kinesthetic information was available. In order to determine the involvement of central processing in retention of these movements we interpolated information-processing tasks of varying difficulty. These tasks were designed to occupy different amounts of S's processing system and were approximately equal in their similarity to the stored information. The results showed that visual information, particularly when it concerned a fixed point in space, showed no loss of accuracy when the interval was unfilled and showed increasing loss as processing difficulty of the interpolated task increased. On the other hand, kinesthetic information generally showed a spontaneous loss of accuracy over time and was little affected by the demands on processing capacity. These results tend to confirm the theoretical analysis outlined above and provide one operational distinction between storage of enactive codes or motor programs and other forms of memory.

One of my reasons for outlining this distinction is that recent data suggest that it may play a useful role in the analysis of some types of brain lesions. Milner (1965a) has reported results from a patient (H. M.) who shows an almost complete inability to learn new things due to bilateral hippocampectomy. However, he appears to show a relatively normal learning curve on mirror tracing and certain forms of manual tracking tasks, despite the fact that he cannot remember having performed these tasks previously. If the effect of hippocampal lesions is upon the central mechanisms that underlie the attention-retention trade-off that I suggested in the last section, it would be expected that they should not affect retention of material that does not place a demand upon this system. Thus H. M.'s ability to learn motor skills would fit this notion. However, other data from Milner's analysis of this patient seems to cast some doubt upon what I have been suggesting (Milner, 1965b; Corkin, 1965). The patient shows a complete inability to learn a maze whether he was visually guided or under kinesthetic control. My colleague, Dr. Keele, however, proposed an explanation for this finding and also an experiment. In the maze task the *S* must correct each error by returning to the last choice point. Thus the movement he makes after an error is in the opposite direction of what he should do. Perhaps these large and frequent corrections tend to disrupt the buildup of a motor program. If the patient were guided through the maze on training trials and then tested at various intervals, this procedure would foster the buildup of an integrated motor sequence that might enable him to learn the maze. We have not performed this experiment, so I cannot say if it would work, but it is interesting to note that H. M. showed an increase in speed as he practiced on the maze even though he showed no improvement in errors,[4] as if he were learning something about how to manipulate the stylus and thus perform the purely mechanical aspects of the task.

It would be a mistake to make too much of a single case or of these rather crude speculations. However, it is possible that more detailed analyses of the formation and retention of motor programs will do much to help us understand the effects of certain types of brain damage.

V. Visual Codes

In the study of visual information codes most of the emphasis has been upon peripheral storage of information prior to naming (Sperling, 1960, 1963). According to some models man recodes all visual input into verbal form in the process of storage. Of course there are compelling reasons to hold that the nervous system can code visual information on a more or less permanent basis without the necessity of verbal labels. Otherwise it would be most difficult to explain such diverse phenomena as delayed matching to sample in nonverbal organisms, eidetic imagery, or the unusually good human ability to retain a visual location in space. The experimental psychologist, however, has been somewhat reluctant to accept this form of storage in man because it is difficult to provide a paradigm in which one can be sure that the retention is visual and not dependent upon a verbal recoding. This is particularly true of the highly verbal college student. Of course, the Sperling (1960) paradigm does this, but it can only be used to describe retention prior to the naming process.

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[4] Professor Brenda Milner described this observation during a visit to our laboratory.

I would like to describe a technique that appears to provide the possibility of investigating many aspects of retention of visual information. The technique depends upon the relatively faster reaction time to physical identity (for example AA) than to name identity (for example Aa), which was discussed in Section II. If the first letter is in memory and the second is presented visually the difference may provide an objective indicant of whether or not *visual* information from the first letter is retained.

In our first study (Posner & Keele, 1967) using this technique, S moved his eyes from the first letter to view a second letter that was available either immediately or after a delay of .5, 1, or 1.5 sec. At zero delay the difference between physical and name identity reaction time was about 80 msec. This difference was reduced to nearly zero if the second letter was delayed 1.5 sec. In other words, the difference initially is about the same as for simultaneous foveal presentation (Posner & Mitchell, 1967), and after about 1.5 sec the advantage of the visual code is effectively lost.

This first study provided evidence for the utility of the RT technique, but the rapid decay of the visual code surprised us. The decay rate seemed remarkably similar to that measured by Sperling (1960). However, there are critical differences in the situations. This was shown rather vividly in a second study (Posner, Boies, Eichelman, & Taylor, 1969). In this study we held the interval between the two letters constant (.5 sec) and varied what was interpolated in that interval. The results showed that a visual masking stimulus (checkerboard) had no effect upon the visual retention of the first letter but that control of central processing capacity (adding a pair of digits) eliminated the visual information. Thus it appears that a visual code defined in this way is not retained in a peripheral storage system like that described by Sperling but, like stored information concerning visual location (Posner, 1967a), requires the availability of central processing capacity.

It may be that the rapid decay rates we observed in the first study were primarily due to the fact that Ss did not choose to try to maintain the visual representation because second letters to be matched were sometimes physically identical and sometimes had only the same name. In our current work we are attempting to determine directly the efficiency with which Ss can retain the visual code of the letter. The most exciting possibility of this technique is that it can be used as an objective test of generation of a visual code. Since a letter is a very familiar figure it is possible to give Ss its name and determine if they can generate a visual representation that will allow them to make a physical match with a subsequent visually presented letter. Work along this line has been encouraging (Posner *et al.*, 1969).

Objective methods for studying rehearsal and generation of visual codes may be of significant aid to the development of analytic accounts of brain damage. Both classic and recent reports of brain deficits (Geschwind, 1965) have indicated deficiencies in imagery. But these studies have entailed introspective accounts that are difficult to analyze. I hope that experimental psychology may be of aid to clinical neurology in providing better techniques of assessment.

VI. Abstraction

In human memory we most often need to deal with complex systems of experience. Individual experiences tend to merge so as to build up an overall impression. The neurologist Henry Head was among the first to recognize the importance of a representational system for storing information that took account of individual perceptions but was not identical with them. Apparently Head developed this view from the writings of Hughlings Jackson, who in turn was heavily influenced by the earlier philosophical notion of abstract ideas (Riese, 1965). Bartlett (1932) introduced the same notion into experimental psychology via his influential book *Remembering*. The process of abstracting the central core of a set of experiences is one that must underlie much of our normal retention (Bartlett, 1932; Oldfield & Zangwill, 1942). It is perhaps to this aspect of memory that the neurologist Goldstein referred when he suggested that his patients had lost the abstract attitude and were unable to rise above the level of concrete instances. More recently, Talland (1965) has pointed to a "premature ending of activation" as underlying the inability of his patients to perform well when the memory trace had to be related to the ongoing experience of the S.

The idea of abstracting a schema or central tendency is at the heart of much psychological theory in the areas of both memory and thinking. However, this theoretical position until quite recently has produced relatively little experimental analysis that might provide an operational basis for its investigation. At present in several laboratories there has been increasing interest in abstraction (Dukes & Bevan, 1967; Evans & Edmonds, 1966; Posner & Keele, 1968). Much of this work has been performed in order to understand how people recognize new experiences based on a series of related past events (pattern recognition; Posner, 1966b; Uhr, 1966).

Consider the set of patterns shown in Fig. 1. The central dot pattern is in the form of a triangle, and the surrounding patterns are statistical distortions of that central figure (prototype). If *S*s are shown a series of

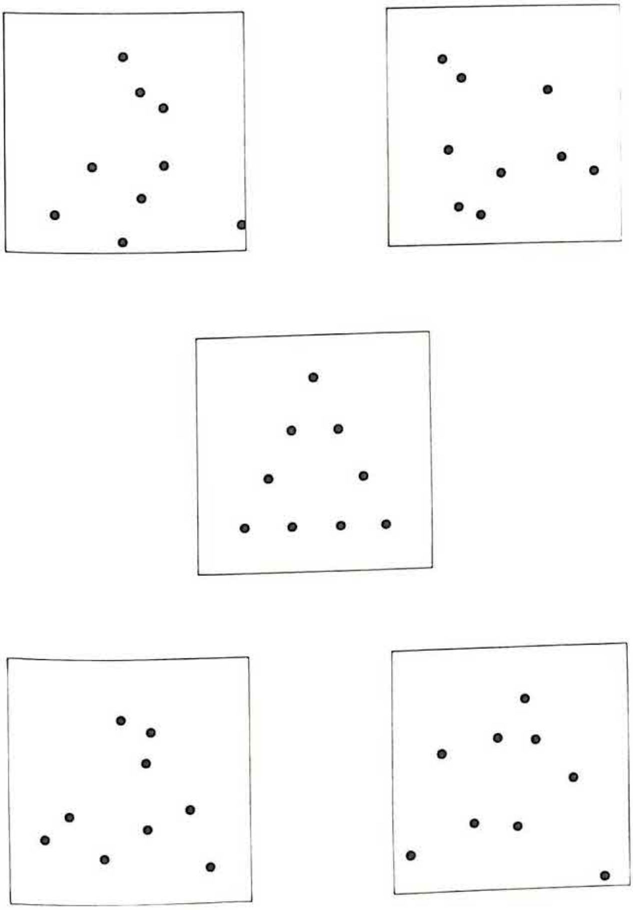

Fig. 1. The central figure is the prototype of a triangle. The four surrounding figures are distortions of the prototype (see text).

these distortions, along with ones that come from other prototypes, they learn to classify the patterns correctly. If they are transferred to new distortions of the same prototypes, which they have never seen before, they do quite well in classifying the new patterns. This process may occur even if *S*s are not informed during learning about what is the correct classification (Edmonds & Mueller, 1967). That is, after seeing a sufficient number of patterns the *S*s can induce the classification for themselves. This also will occur if the prototypes are nonsense patterns rather than familiar figures.

Dukes and Bevan (1967), using photographs, showed that during transfer *S*s do better in recognizing that a new pose represents a familiar face if they are trained on a variety of poses of each face rather than by seeing the same pose over and over again. In a more quantitative way we (Posner, Goldsmith, & Welton, 1967) have shown that higher variability among the instances seen during training facilitates the recognition of new patterns, even if the similarity of the new patterns to those already seen is held constant.

These findings suggest that the human memory system is designed to take into account the fact that experiences with an object do not remain constant. What is called the same stimulus object is different on each trial. In most studies of learning one tries to keep the stimulus situation as rigidly identical on each repetition as possible. This methodology has led us to deemphasize the important advantage of variability of experience, especially for an organism who is almost always going to have to use his learning in a situation that is somewhat different from the condition under which it was acquired.

How do a variety of different individual memories come to be associated in a representation that allows the recognition of new events? To investigate this question we (Posner & Keele, 1968) used a set of three prototype nonsense dot patterns like those shown in Fig. 2. We trained *S*s to learn to associate the four distortions of each prototype with a single response (key press). We then transferred them to a new list of patterns that consisted of the following: the prototypes that they had never seen before, old distortions they had just finished learning, and control patterns at varying distances from the pattern they had seen. We were very impressed with the marked ability of *S*s to recognize the prototype. On the whole they were as good at recognizing the prototype as the patterns they had just finished memorizing and did better than with the other control patterns.

This study seems to provide strong evidence for efficient abstracting ability of the human *S*. Moreover, it indicates that the abstraction process is very definitely tuned to features common to a variety of differ-

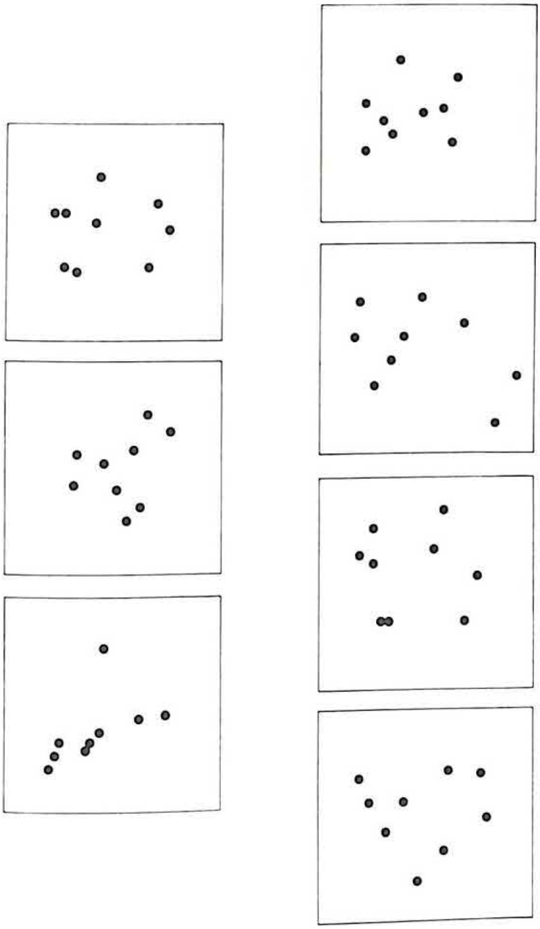

Fig. 2. The three patterns in the left row are nonsense prototypes. The four patterns in the right row are four distortions of the top prototype.

ent patterns. Unfortunately we are almost completely ignorant of the mechanisms that underlie the abstraction process and of the form of representation (visual, verbal, etc.) that is used to store this information. However, present results emphasize that human memory is well designed to abstract from, rather than reproduce, ongoing experience. Perhaps this helps to explain why in the study of human short-term memory use of exact recall always shows memory to be so fragile and subject to disruption (Peterson & Peterson, 1959), while studies of recognition and relearning, which require only a very inexact impression for good performance, indicate human retention to be remarkably tenacious in the face of massive interference from prior and subsequent experience (Shepard, 1967). In any case it is understandable that neurologists should point to impairment in the abstraction ability as a prominent effect of brain damage. Experimental psychology should be able to provide tasks that can give an increasingly precise specification of the components of this ability.

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MECHANISMS OF INTERFERENCE IN FORGETTING[1]

Leo Postman

The interference theory of forgetting was developed in opposition to the classic view that disuse or the sheer passage of time is responsible for progressive declines in retention. The basic postulate of the theory is that forgetting is produced by interference from activities that precede or follow the events to be recalled, that is, by proactive and retroactive inhibition (PI and RI), respectively. A strong law of interference asserts that PI and RI are the necessary and sufficient conditions of forgetting. A weak law of interference limits itself to the proposition that prior and interpolated activities are sufficient conditions of forgetting. The weak law of interference is supported by the large body of experimental evidence for PI and RI. However, whether and to what extent the facts support a strong law of interference is still very much an open question. This question becomes the focus of theoretical interest in the application of the principles of interference to forgetting which occurs outside the laboratory.

In the present discussion we shall consider first some of the principles of interference that have emerged from the results of controlled laboratory studies. Thereafter we shall turn to the application of these principles to extraexperimental forgetting.

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I. Two-Factor Theory

In recent years the two-factor theory of interference, first proposed by Melton and Irwin (1940), has received strong new support from the experimental demonstration of the phenomenon of unlearning. The two factors to which the theory attributes interference at recall are response competition and unlearning of old associations during the acquisition of new ones. The validation of the theory requires independent evidence for the operation of each of the two factors. The occurrence, however infrequent, of overt interlist intrusions provides relatively direct evidence for response competition. It must be recognized, however, that this evidence is not conclusive since it is possible, as Conrad (1960) has suggested, that intrusions are a consequence rather than a condition of the forgetting of correct responses. That is, new responses may become dominant when the old ones cannot be recalled. However that may be, a conclusive demonstration of unlearning calls for a test of recall that can be assumed to be immune to the effects of competition. A procedure that appears to go a long way toward meeting this requirement is the modified free recall test (MMFR) introduced by Barnes and Underwood (1959). The subject, after having learned two successive lists conforming to the A-B, A-C paradigm, is presented with the common stimulus term A and asked to recall both responses in the order in which they occur to him. The effects of competition are assumed to be minimized because the test is unpaced, so that a momentarily dominant response need not displace a weaker one; if both are available, both can be given. Moreover, failure of differentiation is eliminated as a source of error because both responses are called for and identification of list membership is not required.

It is important to bear in mind that the theoretically critical properties of the test are postulated on *a priori* grounds. The assumption that the effects of competition are minimal is predicated upon the conception of competition as the momentary dominance of one associative response over another or the equally transitory mutual blocking of two responses of approximately equal strength. Even with respect to this conception troublesome problems of definition come to light when one examines the assumption that the MMFR test is free of the effects of competition. The question arises of how long a period of dominance or blocking must last before it becomes a loss of availability. The line between these two classes of events is blurred on a self-paced as well as on a paced test. Probably more important, when other possible forms of competition are considered, for example, competition between alternative repertoires or systems of responses, there is no longer any *a priori* reason to assume that modified free recall is immune to the interferences they may produce. Thus the possibility must remain open that MMFR tests do not provide pure estimates of unlearning as defined in the original statement of the two-factor theory.

If one grants for the moment the properties attributed to the MMFR test, there is by now substantial evidence for unlearning in the sense of reduced availability of first-list responses at the end of interpolated practice. Given this evidence, two major questions of interpretation arise: (1) What is unlearned? (2) What are the events during the acquisition of the interpolated material that are responsible for unlearning?

What is unlearned? A widely accepted answer to this question is given in an analysis by McGovern (1964). For paired-associate learning, this analysis specifies three classes of associations that are subject to unlearning: (1) forward associations, (2) backward associations, and (3) contextual associations. Forward and backward associations link the stimulus and response members of the prescribed pairs. We shall consider these together under the heading of specific associations. Contextual associations refer to the connections established between the response terms and the environmental context, for example, the cues provided by the room and the experimental equipment. The development of contextual associations is assumed to facilitate response recall which is an essential step in the mastery of the paired-associate task. With this analysis of associative components as a point of departure, McGovern proposed that there would be unlearning of the first-list component whenever the A-B, A-C paradigm (old stimuli and new responses) applied to that component during second-list learning. Thus, when the successive lists conform to the A-B, C-D (new stimuli and new responses) paradigm, only contextual associations are unlearned; in the A-B, A-C paradigm both contextual and forward associations are subject to unlearning, and so on.

While it has been possible on the basis of this analysis to predict the relative amounts of RI under various conditions of transfer, this conceptualization of the components of unlearning may not prove satisfactory in the long term. In particular, the explanatory power of the concept of contextual association is open to serious question, both on *a priori* and empirical grounds: (1) Contextual associations have reference to the connections that develop between features of the physical environment and the repertoire of responses to be recalled. It is likely that such physical features have much less weight than the verbal environment in defining the context to which the members of a given set of responses become associated. (2) A summation of independent habits during acquisition and a corresponding subtraction of habits during unlearning are implied. Available analyses fail to support the assumption that habit summation occurs in verbal learning (for example, Hill & Wickens, 1962). (3) Most important, there is substantial evidence that unlearning, as measured by MMFR tests, is largely a matter of reduced response availability. It is a fact that RI develops more slowly and remains at much lower levels when retention is measured by tests of associative matching or recognition rather than MMFR (Garskof & Sandak, 1964; Postman, 1965; Postman, Stark, & Fraser, 1968; Sandak & Garskof, 1967). For example, under conditions of massive interpolation which produce losses of 50% or more in MMFR, the percentage of RI on a test of associative matching is typically of the order of 10–15. These differences are obtained when response recall is required during the acquisition of the successive lists, for example, when the method of anticipation is used. When the requirement of response recall during acquisition is eliminated by use of a multiple-choice procedure, the amount of RI is reduced further. Thus, S's ability to discriminate between correct and incorrect first-list associations remains largely intact after a high degree of interpolated learning (Postman & Stark, 1969).

Within the framework of current assumptions about the components of unlearning one would have to conclude that the losses observed in MMFR are in large measure attributable to the extinction of contextual associations. Such a conclusion immediately raises some serious interpretative problems. First, there is no ready theoretical explanation for the apparent finding that contextual associations undergo extinction far more rapidly than do specific ones. Response learning must precede the establishment of specific associations; moreover, specific associations should be more subject to intralist interference than contextual associations. On *a priori* grounds, therefore, contextual associations would be expected to be stronger and more resistant to extinction. Second, internal inconsistencies come to light when an assessment of the components of unlearning is made for various paradigms of transfer. The difference between the amounts of RI obtained in MMFR and on tests of associative matching is greater for the A-B, A-C than for the A-B, C-D paradigm. Yet the shifts should be of the same order of magnitude since the inferiority of MMFR performance is attributed to the loss of contextual associations, and that loss should be equal for all paradigms in which the successive responses are new and unrelated.

In sum, the empirical answer to the question of what is unlearned is that the availability of old responses is rapidly and substantially reduced during the acquisition of new ones but that there is a relatively slow and limited decline in the retention of specific associations. This pattern of results is not readily deducible from the distinction between contextual and specific associations.

We now turn to a consideration of the mechanisms of unlearning, that is, the nature of the events during interpolated practice or at the time of the retention test that produce the observed losses in performance. The prevailing view is still that originally proposed by Melton and Irwin (1940), viz., that unlearning is analogous to extinction. The unreinforced elicitation of the old responses during the interpolated activity, as in the A-B, A-C paradigm, is regarded as the necessary and sufficient condition of unlearning.

The most direct index of the frequency of such unreinforced elicitations, viz., the number of overt first-list intrusions during second-list learning, is not correlated with the amount of RI (for example, Keppel & Rauch, 1966). This fact is not conclusive, however, if the observed frequency of intrusions merely reflects changes in the ratio of overt to covert errors. On this assumption there are several experimental findings that appear to give at least indirect support to the view that the amount of unlearning is significantly related to the frequency with which the earlier responses are elicited as either overt or covert errors during the acquisition of subsequent tasks. In the interest of brevity only one illustrative finding will be mentioned here. There is significantly more unlearning when the class of responses (for example, letters or adjectives) in the successive lists conforming to the A-B, A-C paradigm remains the same than when it changes (Postman, Keppel, & Stark, 1965). It is reasonable to suppose that the probability of unreinforced elicitations varies directly with the similarity of form class of the competing responses. The frequency of overt interlist intrusions, although low as usual, is in accord with this assumption. It may be noted that this finding adds to the evidence that, contrary to the principle of Osgood's (1949) surface, interlist interference increases as a function of response similarity.

If it is granted that unreinforced elicitations of the old responses are significantly related to unlearning, two alternative hypotheses can be entertained: (1) Unreinforced elicitations constitute the mechanism of unlearning and are in themselves responsible for the observed losses in availability. This is the essence of the extinction analogy. (2) Unreinforced elicitations are but a link in the chain of events that result in unlearning, that is, they trigger other mechanisms which in turn are the effective determinants of losses in availability. In developing an argument for the second alternative I shall take my point of departure from the fact that as a result of interpolated learning response recall declines much more rapidly than does the retention of specific associations. Since responses to individual stimuli are assumed to occur as errors and to undergo extinction, the specific stimulus-response associations should be progressively weakened. In fact, however, it is the responses which are unlearned rapidly, whereas specific stimulus-response associations are relatively resistant to interference.

These considerations lead to the supposition that the mechanism responsible for unlearning operates primarily if not exclusively on the entire repertoire of first-list responses rather than on individual stimulus-response associations. Specifically, the hypothesis suggests itself that unlearning is a consequence of the operation of a mechanism of response selection under conditions of negative transfer.

The operation of a selector mechanism was first inferred by Underwood and Schulz (1960) from the highly dependable tendency of subjects learning a verbal list to restrict their overt responses to items from within that list. Furthermore, when there is a change in required associations, as in a transfer task, the subject comes quickly to restrict himself to the newly appropriate repertoire. He appears to adopt what has been described elsewhere (Postman, 1966) as a rule of response restriction appropriate to the paradigm of transfer. The rule may specify the newly correct responses in terms of their relation to the old ones, as in A-B, A-B′ (old stimuli and similar responses), or it may prescribe the substitution of new responses for the old ones, as in A-B, A-C. Criteria of selection consonant with the appropriate rule of response restriction begin to regulate the subject's responses immediately upon exposure to the transfer task, as is apparent from the low frequency of overt interlist intrusions violating the rule. They may not, however, prevent the occurrence of covert intrusions.

It is likely that the selector mechanism is as effective as it is because from the onset of practice on a given list the relative recency of occurrence of the currently prescribed items provides a ready basis for the arousal of the appropriate responses. As Underwood and Schulz put it: "The subject responds only with items or letters from which he has received recent stimulation. The mechanism in the present situation is 'set off' by the responses acting as stimuli, the result being that only the recall strength of those responses . . . is activated (1960, p. 144)." Relative recency, of course, is not the only potential basis for the delimitation of the ensemble of responses. Formal and semantic similarities among the items in the current repertoire which distinguish them from items in the earlier list likewise will serve to facilitate the operation of the selective process. In the acquisition of successive tasks, relative frequency of occurrence in the situation provides an additional basis of discrimination between appropriate and inappropriate responses (see Ekstrand, Wallace, & Underwood, 1966).

Relative recency and frequency are fallible criteria of selection. Only when the successive repertoires are highly distinctive in other respects can the mechanism of response selection function with maximal efficiency so that first-list associations are unlikely to be elicited as errors during second-list learning. Such appears to be the case when the responses in the two lists are from different form classes (Postman *et al.*, 1965). However, failures of selection will occur when the responses in the two lists are drawn from the same pool, especially when the stimuli remain identical (A-B, A-C) and provide a common verbal context. As a consequence, first-list associations will be elicited as covert or overt errors. If the continued occurrence of such errors is to be prevented or reduced, the criteria of selection must become increasingly restrictive so as to exclude the intrusion of previously correct but now inappropriate responses. The development of such highly restrictive criteria of selection is equivalent to the suppression of earlier responses. The heavier the interference from the latter, that is, the greater the negative transfer, the more nearly complete will be the suppression of the conflicting repertoire. It should be emphasized that the mechanism of selection, and the consequent suppression of sources of interference, are seen as operating on ensembles of responses, for example, the first-list and second-list repertoires, rather than on individual associations.

According to the present interpretation, the reduced availability of first-list responses immediately after the end of interpolated learning reflects in large measure the persisting dominance of the most recent criteria of selection and hence the continuing suppression of the earlier repertoire. At this point in time a shift back to the criteria of selection established during first-list learning is difficult and can be effected only intermittently. Hence only the strongest first-list responses are likely to be re-aroused and recalled. However, the dominance of the most recent criteria of selection may be assumed to diminish over time. Thus, as the suppression is gradually lifted there should be recovery of the first-list repertoire. Evidence in accord with this expectation was obtained in a recent series of studies on temporal changes in interference (Postman *et al.*, 1968).

II. Proactive Inhibition

According to classic two-factor theory, RI represents the combined effects of unlearning and competition, whereas PI is attributed to competition alone. On the assumption that the MMFR procedure minimizes or eliminates the effects of competition, performance on such tests should be free of PI. Contrary to this expectation, however, several experiments have yielded evidence of substantial PI when retention was tested by MMFR (Houston, 1967; Koppenaal, 1963; Postman *et al.*, 1968). From the assumptions of the theory it follows that the MMFR test does not in fact render performance immune to the effects of competition. If the conclusion is accepted, the type of competition has to be specified. Our analysis of the mechanism of unlearning suggests that the effective competition is not a matter of the momentary dominance of one response (C) over another (B), both of which have been attached to a common stimulus (A). Rather, the interference is between the two systems of responses acquired in the successive tasks. When two lists conforming to the A-B, A-C paradigm have been learned, the mechanism of response selection has operated on two repertoires, one comprising the B terms and the other the C terms. After the end of interpolated learning, as the dominance of the most recent criteria dissipates, the first as well as the second repertoire is liable to activation. The simultaneous activation of two (or more) response systems creates a condition of interference at recall. The effective competition is assumed to be generalized (see Newton & Wickens, 1956), that is, to exist between response repertoires rather than between conflicting associations to a given stimulus. Hence the detrimental effects on recall are observed on unpaced as well as on paced tests.

The fact that PI increases as a function of time is consistent with this assumption, since discrimination between the alternative response systems should become more difficult as the retention interval is lengthened. Hence conditions that serve to maintain the long-term distinctiveness of alternative response systems should produce a reduction in PI. In accord with this expectation the amount of PI is minimized if not eliminated when the interfering list is learned by distributed practice and the test list by massed practice (Underwood & Ekstrand, 1966). The difference in the temporal distribution of learning trials provides a continuing stable basis of discrimination between the two response systems. By contrast the sheer degree of learning of the interfering task has little if any effect on PI. The fact that it is the distinctive temporal characteristics of the tasks which are critical is supported by the further finding that the beneficial effects of distribution are largely eliminated when the interfering list and the test list have a number of pairs in common (Underwood & Ekstrand, 1967a). The presence of the repeated pairs invalidates the basis of differentiation between the two tasks, viz., the distribution of learning trials on the two types of materials.

III. Departures from Two-Factor Theory

It may be useful at this point to underscore the differences between the present analysis and the traditional formulation of two-factor theory. First, major emphasis is placed on the distinction between response loss and the forgetting of specific stimulus–response associations. Response loss per se is assumed to reflect the suppression of an initially correct but subsequently inappropriate repertoire of responses. The possibility is left open that specific associations which are persistent sources of error are weakened or suppressed, but the latter process is at most slow and gradual. Second, the parallel distinction between generalized and specific response competition is stressed. Generalized competition is between alternative response repertoires; thus a persisting tendency to suppress the first-list repertoire on a test of recall constitutes a powerful source of generalized response competition. In addition, there may be competition between specific responses to a given stimulus, that is, reproductive inhibition in the classic sense (McGeoch, 1942), but the evidence for the latter is far from compelling. Third, response suppression is viewed as a partially reversible process so that there is a progressive increase in the availability of first-list responses or apparent spontaneous recovery. Finally, with the simultaneous activation of alternative response systems, generalized competition produces proactive inhibition on unpaced as well as on paced tests of recall.

IV. Extraexperimental Sources of Interference

We now turn to the application of the principles of interference derived from controlled laboratory studies to forgetting that occurs outside the laboratory. The basic fact which requires explanation is that retention declines as a function of time even in the absence of formal interfering activities. A naive *S* who learns a single list in the laboratory to a criterion of one perfect recitation will typically show a loss of 15-20% after 24 hours. To what factors can this retention loss be attributed? The strong law of interference—the principle that habit interference is a necessary and sufficient condition of forgetting—entails the conclusion that the retention loss results from retroactive and proactive inhibition produced by experiences outside the laboratory. According to a weak law of interference, interference from extraexperimental sources may be a condition of forgetting but does not necessarily account for all retention losses. A decision between the strong law and the weak law would be difficult at best because it would require a determination of whether interference effects account exhaustively for all the observed variations in forgetting. However, both the strong and the weak position at the very least must be supported by a demonstration that extraexperimental interferences significantly influence the course of forgetting.

In the forgetting of verbal materials learned in the laboratory, language habits acquired or practiced before or after the acquisition of the critical task are highly probable sources of extraexperimental interference. On *a priori* grounds proactive interference from extraexperimental sources is likely to exert a considerably greater influence than retroactive interference. Language habits have been acquired and practiced throughout the individual's lifetime prior to the acquisition of the experimental task, and on this time scale the retention intervals typically used in experimental studies are quite short and afford only limited opportunities for the development of habit interference.

Two classes of prior language habits that are potential sources of extraexperimental interference are (1) letter-sequence habits and (2) unit-sequence habits (Underwood & Postman, 1960).

Letter-sequence habits reflect the sequential probabilities of letters in the words of the language. Letter-sequence interference will occur to the extent that the sequences of letters prescribed in the experiment deviate from those characteristic of the language. That is, the lower the probability of the prescribed sequence in the language the more interference there should be from conflicting sequences at the time of recall.

Unit-sequence habits are based on the sequential dependencies of integrated verbal units such as words; thus the subject is assumed to come to the laboratory with a hierarchy of associations to each of the verbal units included in the learning task. It was predicted that the amount of unit-sequence interference in the retention of new verbal associations would increase directly with the frequency of occurrence in the language of the words to be learned. Normative studies show that the number of different associates elicited by a word per unit of time increases with the frequency of usage; it is reasonable to suppose that the same relation holds for the strength of associations. When a subject is required to learn an arbitrary verbal sequence, the number and strength of competing associations, therefore, should increase with the frequency or meaningfulness of the words. The resulting interference should be especially heavy if preexperimentally associated items appear within the same list but not in the appropriate order, for example, if *table* and *chair* both are in the list but do not follow each other. Under these circumstances prescribed and competing associations cannot be differentiated on the basis of the list membership of component units. In summary, two gradients of extraexperimental interference may be specified: the retention of letter sequences should vary directly with the probability of sequences in the language, whereas the retention of word sequences should be related inversely to the frequency or meaningfulness of the items.

Tests of these theoretical predictions call for comparisons of the rates of forgetting, with degree of learning held constant, of the classes of materials specified in the analysis of potential sources of interference—probable vs improbable letter sequences and high-frequency vs low-frequency words. With a few exceptions (for example, Turnage, 1967), the results of the relevant experimental studies have failed to bear out the theoretical predictions (Ekstrand & Underwood, 1965; Postman, 1961, 1962; Underwood & Ekstrand, 1967b; Underwood & Keppel, 1963). In general, the amount of forgetting over intervals of up to a week has been found to be essentially the same for materials of high and of low meaningfulness if the degree of original learning is carefully controlled. It appears, in fact, that degree of learning is the only variable that produces dependable major variations in the amount of forgetting. The conclusions thus far are essenially the same for letter-sequence and unit-sequence interference. The discussion which follows will be limited to the problem of unit-sequence interference both because it has been studied more extensively than letter-sequence interference and because it provides a better point of departure for consideration of the problems of method and interpretation posed by the apparent empirical failures of the theory.

In spite of the dearth of supporting evidence, there has been some reluctance to abandon the theoretical conceptions underlying the hypothesis of extraexperimental sources of interference. There are two major reasons for this reluctance. First, interference theory receives strong support from the phenomena of retroactive and proactive inhibition observed in the laboratory. It does not seem likely that there is complete discontinuity between the phenomena of forgetting studied in the laboratory and those which occur outside the laboratory. Second, there is no well-developed and experimentally supported alternative to the principles of interference as an explanation of long-term extraexperimental forgetting. Historically, the major alternative view has been decay theory, which attributes forgetting to the progressive degradation of memory traces over time. However, it has been extremely difficult to devise conclusive tests of the decay hypothesis (see Postman, 1964). Moreover, the facts of retroactive and proactive inhibition are not easily reconciled with decay theory. Clearly the passage of time does not in and of itself determine the course of forgetting; the nature of the activities preceding and following the critical task can be shown to be decisive, at least in the laboratory. In the light of such considerations a considerable amount of effort is being devoted to the analysis of the reasons for the apparent failures in the application of interference theory to extraexperimental forgetting.

We shall focus on two types of analysis that bring out the shortcomings of the theory and may help to throw light on the empirical failures. One approach has been to reexamine the relations between language habits and the requirements of the experimental tasks, with special emphasis on sources of facilitation as well as of interference. The question is raised of whether in predicting a direct relation between meaningfulness and forgetting the unit-sequence hypothesis fails to take adequate account of the positive as well as of the negative effects of preexperimental habits on retention. Another approach is to consider the question of whether the operations that have been used in the experimental analysis of retroactive and proactive inhibition are representative of the conditions of interference that obtain outside the laboratory. If they are not, then predictions about extraexperimental forgetting must be reassessed by means of procedures that more nearly duplicate conditions outside the laboratory.

Does the unit-sequence hypothesis fail to take adequate account of the positive effects of preexperimental habits on retention? For purposes of considering this question it will be useful to focus again on the two components of forgetting that were distinguished in the analysis of unlearning: reduction in the availability of responses and associative loss. The prediction of a direct relation between frequency of usage or meaningfulness and forgetting clearly refers to the component of associative loss; the number of potential competitors at recall is expected to increase with the meaningfulness of verbal units. Consider now the component of response recall. There are reasons to believe that the effects of frequency or meaningfulness on this component are positive rather than negative. It is known that (1) the number of preexperimental associations within a set of high-frequency words is greater than within a set of low-frequency words (Deese, 1959) and (2) that such interitem associations facilitate free recall, that is, they have a positive effect on the availability of the responses when the items do not have to be given in any particular order. The effects on response availability are positive because the interitem associations restrict the range of alternative responses, particularly if these associations reflect conceptual or categorical groupings of items.

Interitem associations thus have dual and opposed effects: They favor response recall, but they interfere with the reproduction of serial order when that order is arbitrary and does not conform to preexperimental sequential habits (see Weingartner, 1963). The net effect will depend on the requirements of the retention test, and it may also change with the length of the retention interval. As the interval between learning and test is lengthened, the factor of response recall may assume increasing importance relative to the interference from conflicting associations. Moreover, the experimental evidence we reviewed earlier suggests that specific associations are relatively resistant to forgetting. Thus, when differences in interitem associative strength are maximized, for example, when serial lists of conceptually related items are compared with lists of unrelated words, the effects on response availability are sufficiently great to give the related lists an advantage even in long-term ordered recall (Postman, 1967).

It now appears that the original predictions regarding the relation between meaningfulness and retention were an oversimplification because they failed to take account of the divergent effects of language habits on response recall and on the retention of sequential associations and of the covariation of sources of facilitation and interference. To the extent that there is independent evidence for the operation of these component processes, observed measures of retention may be viewed as representing the balance of positive and negative effects of prior language habits peculiar to a given test situation. It would be premature to conclude, however, that all previous failures to confirm the theoretical predictions reflect a near balance of positive and negative effects, although in each case this possibility must be considered seriously.

Are the conditions of interference studies in formal experiments on retroactive and proactive inhibition representative of the conditions which obtain outside the laboratory? In a recent study Underwood and

Ekstrand (1966) pointed to two important reasons for questioning the representativeness of laboratory studies of interference. First, language habits are obviously of considerably greater strength than interfering associations established in the laboratory. Second, language habits are acquired under conditions of widely distributed practice, that is, successive occasions of practice are separated by varying intervals of time. It appeared possible, therefore, that one or both of these differences between experimental and extraexperimental conditions are responsible for the apparent discrepancy between the laws of retroactive and proactive inhibition on the one hand and the laws of extraexperimental forgetting on the other. To test this hypothesis Underwood and Ekstrand attempted to simulate in the laboratory the characteristics of proactive interference (assumed to be the more important mechanism of long-term forgetting) that hold true for extraexperimental associations. Thus, two variables were manipulated in a study of proactive inhibition, viz., degree of learning and distribution of practice on the interfering prior list. As already noted, the amount of proactive interference was strikingly less when prior learning was distributed than when it was massed, but there was no reliable effect of the degree of prior learning on the retention of the test list. Finally, an MMFR test showed virtually perfect recall of the prior list when it was learned by distributed practice.

The results of Underwood and Ekstrand thus raise the serious possibility that preexperimental habits are less effective than laboratory habits in producing interference at recall because their acquisition is widely distributed over time. Exactly why the factor of distribution serves to reduce interference cannot be said with certainty. It already has been suggested that a difference between practice schedules provides a basis for highly accurate discrimination between alternative response repertoires, for example, between experimental associations and other verbal sequences. This interpretation again brings to the fore the importance of temporal cues in maintaining the distinction between appropriate and inappropriate responses.

It must be recognized that the application of the principles of interference theory to extraexperimental forgetting frequently has resulted in empirical failures of the predictions. Some of the possible shortcomings of the theory are beginning to be explored systematically and may account for the discrepancies between predictions and empirical findings. However, we are still a long way from having an adequate explanation of the retention losses which are regularly observed over time and the magnitude of which appears to be largely independent of the nature of the learning materials.

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ITEM RETRIEVAL AND ORGANIZATION IN RECALL

Charles N. Cofer

My invitation to participate in this symposium is based, I presume, on the fact that for several years much of my research has been devoted to the study of free recall and to factors involved in its organization. In this paper I shall first outline the general character of work on free recall and then become somewhat more specific concerning the role of free recall in the study of memory and make some comments about its possible usefulness to the study of deranged memory.

As a method, free recall has a long history (Tulving, 1968), but it has become prominent only in the past 15 years, since Bousfield (1951, 1953) described the procedures. We can distinguish two major procedural variations employed in free recall and perhaps three major problems to the study of which they have been devoted.

I. Procedural Variations

Bousfield (1953) mainly has employed the procedure in which a single presentation of a long list of words, usually 40 or more, is followed by having the subject recall as many of those words as he can, *in any order in which they occur to him.* The words are typically presented one at a time, aurally or visually, and it is the freedom to recall the items in any order that distinguishes free recall from other procedures such as serial recall. The other major method has been called free recall learning (Tulving, 1962) or free learning (Eckstrand & Underwood, 1963). In essentials, it is simply multitrial free recall, that is, there are several trials, each one consisting of a presentation of the list of words (usually in different orders from trial to trial) followed by free recall of the items.

II. Problems Investigated

As to the problems studied, it is convenient to classify them in three ways:

1. Response learning. Since in free recall the response items are not paired or associated with any particular stimulus, it has been suggested that the technique can be used to measure the learning of a set of responses independently of their associations with particular stimuli. This has permitted direct investigation of the response learning stage, as distinct from the associative stage, for example, in paired associate learning (see Underwood & Schulz, 1960). I shall not be concerned with this usage here.

2. Characteristics of the verbal repertoire. Since Bousfield's work (1953) and that of Jenkins and Russell (1952), it has been known that in the free recall of a list of words subjects will organize the items at recall in a manner different from the way they were organized at presentation. Bousfield used lists of words the members of which belong to four mutually exclusive categories, such as animals, vegetables, articles of clothing, and items of furniture. Jenkins and Russell incorporated pairs of words in their lists, the pairs consisting of stimuli from the Kent-Rosanoff word association test and normatively frequent responses to these stimuli. In both cases, for the categorized items and the associated items, the words were arranged in a random order for presentation. In their recall, however, the subjects tended to cluster together the categorized or the associated words to an extent in excess of chance expectation. These tendencies have been termed "category clustering" and "associative clustering," respectively. Interword relations that lead to clustering of either type may be said to exemplify characteristics of the verbal repertoire. That is to say that the relations among the words in the list at presentation must exist in the subject's verbal organization if he organizes the randomly presented items in accordance with those relations in his recall. Some work has been

done that indicates that associative clustering is a function of normative free association strength (Jenkins, Mink, & Russell, 1958; Marshall, 1967) and that a number of category relations are reflected in clustering in recall (Tulving, 1968). There are cases, too, in which logical relations in the language, such as synonymity (Cofer, 1959), mediate little clustering or in which other relations, like that of form-class, mediate no clustering (Cofer & Bruce, 1965). To my knowledge, however, no study has attempted an exhaustive charting of the organization of the verbal repertoire by means of clustering in free recall.

Without using clustering, Deese (1959) has shown that interword associations are highly related to the total recall scores for lists of 15 words.

3. Organization and recall. From the first it has been suggested that item recall is greater when the words of a list are interrelated in some way than when they are not interrelated. I have summarized the surprisingly meager evidence on this point elsewhere (Cofer, 1967, pp. 182–183). It is generally believed that the presence of the interword relations facilitates the organization of the material and that this organization abets or facilitates item retrieval. Many of the studies of free-recall learning have been motivated by this notion and have supported it (Tulving, 1962, 1964).

In the case of single-trial free recall, however, the interpretation of the augmentation of recall for related words as against unrelated words is not entirely clear, at least to me. There are two interpretations that may be offered. One of them is that the interword relations provide, somehow, for a better "trace" of a list of related words than for a list of unrelated words. To put this differently, there is more learning when the items are related than there is when they are not related, and the augmentation of recall reflects this augmented learning.

The other interpretation is that the same number of words is "learned" in the case of lists of both kinds and that the superior recall for the lists of related items arises from the successful "generation" of items from those actually recalled from a list of related words. The interword relationships may permit this generation of items that cannot readily be done when there are no interrelations among the items. Over the past several years I have accumulated some evidence which, it seems to me, supports the notion of generation. A supplement to generation is required, however. This involves the ability of subjects to discriminate among those items they generate as to which were on the list and which were not.

III. Evidence on the Generation-Discrimination Hypothesis

There are some boundary conditions that ought to be specified so far as this tentative conclusion is concerned. The kind of situation to which I will apply the generation and discrimination hypothesis is as follows: A list of words, usually 40 of them, is presented a single time, either aurally or visually, at a 2-sec rate. The subject knows at list presentation that he is to recall the items in any order in which they occur to him, and immediate, written recalls are obtained. Of course, there are variations in these conditions that I will specify as needed. The words of the list typically belong to one of four mutually exclusive categories, for example, 10 words each to such categories as animals, vegetables, occupations, and articles of clothing. The recall protocols are examined for number of items correctly reproduced, for intrusions, and for clustering; clustering is measured by the Ratio of Repetition or RR. This measure is defined as the number of repetitions of items from the same categories less the number of clusters, divided by the number of items recalled minus one.

Observations that directed my interest to the relationship between organization and item recall were those that suggest that variations in organization (RR) are not always associated with variations in the same direction for item recall. In a number of experiments I have obtained two recalls from the same subjects following a single presentation of the list, the two recalls being separated by a 5-min filled interval. In 38 such comparisons, RR increased between recalls 37 times, whereas number of items recalled increased in only 12 instances. Another piece of evidence came from experiments by Cofer, Bruce, and Reicher (1966). They used two kinds of lists and two types of presentation. In both kinds of lists there were four sets of ten categorized items each, but in one list the items were high-frequency associates of the category names whereas in the other list the items were low-frequency associates of the same names. One method of presentation was a random order, but the other one was a blocked method in which all the items from one category were presented one after another, then the items from another category, and so on. RR was augmented by blocked presentation for both lists, and item recall was also increased for the list of high-frequency associates. However, item recall did not differ for the low-frequency list with method of presentation at any of the three exposure intervals used. Here again, for the low-frequency list, there is a clear discrepancy between RR and item recall. I could cite other evidence on this point, but what I have reviewed perhaps is enough to indicate that there is no necessary correspondence between organization (RR) and item recall.

The differences in items recalled that are obtained between lists of related items and lists of unrelated items still remain, of course, and the processes responsible for the differences require identification. One technique I have used seems to offer some promise in this regard. I have called the technique forced recall, although I think now that this is a misnomer. It consists in obtaining, in the usual way, free recall of the lists presented and then requiring the subject to produce enough additional items to match list length. My interest has focused on the kinds of items added under forced recall.

Let me describe several experiments using the method of forced recall, the major variations among the experiments being the lists employed. In one experiment, carried out by Miss Bonnie Travellet, two lists, one composed of high-frequency and the other of low-frequency associates of the category names, were used. For one list of each kind the items were presented in a random sequence and, in the other, in a blocked sequence. Free and forced recalls were obtained, and the recalls were analyzed for items correct, RR, relevant intrusions (that is, items not on the list but members of the list categories), and irrelevant intrusions. The data for forced recall are shown in Table I. [In regular free recall, the findings of Cofer *et al.* (1966), who used the same lists and procedures, were replicated.]

Table I

Forced Recall Data from Travellet's Experiment

	Number items in forced recall	Mean percent "correct"	Mean percent relevant intrusions	Mean percent irrelevant intrusions
High frequency, block	273	26.0	48.0	26.0
High frequency, random	314	29.0	42.0	29.0
Low frequency, block	386	8.5	71.0	20.5
Low frequency, random	415	7.5	56.5	36.0

Table I shows that for the high-frequency lists a substantial proportion of the items added during forced recall would have been correct had they occurred in free recall. This is not the case with the low-frequency lists, for which, on the other hand, the proportion of responses added in the relevant intrusion category is higher than it is for the high-frequency lists. Note also that a substantial proportion of the added items are irrelevant intrusions. Two other experiments, carried out by Howard Walker, show substantially the same results. In these experiments nouns were presented alone as well as in adjective-noun pairs. Different lists were used in these experiments, and in the first one a blocked presentation procedure also was utilized. Table II shows the results of the first experiment. Again, blocked presentation of nouns alone augments the proportion of relevant intrusions, but with this list does little for number "correct." Modification, with or without block presentation, appears to depress relevant intrusions. In Walker's second experiment, the results for forced recall of which are shown in Table III, for one of the groups presented (nouns alone) the categories of the list were identified for the subject prior to list presentation. This maneuver augmented, as did block presentation in Travellet's experiment, both added "correct" responses and relevant intrusions. Again, modification suppresses relevant intrusions.

Table II

Forced Recall Data from Walker's First Experiment[a]

	Mean percent "correct"	Mean percent relevant intrusions	Mean percent irrelevant intrusions
Nouns only, block	8.69	36.62	54.70
Nouns only, random	7.06	22.58	70.36
Modified nouns, block	5.24	14.69	80.07
Modified nouns, random	7.24	15.56	77.10

[a] The values are percentages of total responses classified into each kind of forced recall response.

Table III

Forced Recall Data from Walker's Second Experiment[a]

	Mean percent "correct"	Mean percent relevant intrusions	Mean percent irrelevant intrusions
Nouns alone	9.6	21.7	68.7
Nouns alone, categories identified	16.8	41.9	41.3
Adjective-noun pairs	7.0	8.4	84.6
Noun-adjective pairs	3.2	8.1	88.7

[a] The values are percentages of total responses classified into each kind of forced recall response.

The data from these three experiments give a fairly clear-cut picture. With lists whose members are high-frequency associates of the category names, a quarter of the responses added in forced recall are "correct" and about half are relevant intrusions. For low-frequency lists, however, correct responses added seldom exceed more than 10%. Depending on the list and other conditions, relevant intrusions may or may not constitute a large proportion of the remaining responses added in forced recall.

These results resemble those obtained with the forced recall procedure in earlier experiments carried out with 15-item lists (Cofer, 1967). These lists varied in interitem associative strength (Deese, 1959), that is, the lists were composed of items which tend, in free association, to elicit each other as responses to varying degrees (including zero). The number of correct responses generated in forced recall to these lists correlated at 0.73 with the index of interitem associative strength. However, the number of such correct responses was not large, never exceeding a mean of one response. The analysis made of the remaining forced recall responses was in terms of whether the responses could or could not be identified as free association associates of the list name or of list members. If we impose the restriction that a response is an associate only if it occurs in free association as an associate to *both* the list name and one or more list members, the proportions of such responses are as follows: high-frequency list, 15%; low-frequency list, 38%; zero-frequency list, 5%. This pattern of results resembles those for relevant intrusions with high- and low-frequency categorized lists.

From the data I have presented so far, it seems that the effect of categorization or of interitem associations is to focus the subject's responses on items that are relevant to the list; he tends to produce more relevant intrusions for low-frequency lists, categorized or interassociated, and fewer correct responses than he does for high-frequency lists. The interesting point is that if these relevant intrusions are available, as forced recall indicates them to be, why are they not produced in free recall? If we can find a reason for their failure to occur in free recall, can we then apply this reason to the apparently greater recall of lists of related than of unrelated words?

One possible reason that available items may not appear in free recall is that subjects will record only items that they were reasonably certain were on the list. In a direct investigation of this possibility I asked subjects to give free recalls of 15-item lists but in doing so to be sure to match list length. A code was provided so that the subject could record, for each item he wrote down, whether he was sure it was on the list, sure it was not on the list, or uncertain about its status as a list member. Over six lists, of varying interitem associative strengths, the word was correctly identified when it had been on the list on the average of 93% of the time; when the words were not on the list they were correctly so identified, on the average, 69% of the time. This evidence suggests to me that subjects are wary of putting items into their recalls unless they are reasonably certain the words were actually presented. Subjects, by the way, manifest a considerable reluctance to add enough items to match list length. I might add that it is a widely known fact that extralist intrusions are rare in paired-associate and serial learning situations, as well as in free recall.

The evidence I have presented so far permits the following conclusions: with lists of categorized or interassociated items, when required to do so, subjects can generate items relevant to the categories or to the associative networks represented. However, they will not write down such generated items unless they have reasonable certainty—or recognition memory—that the items were on the list. With high-frequency lists, of course, the items generated, being high-frequency associates, will tend to have been on the list. Recognizing this, the subject can record them, not because he has recalled them but because having generated them he can recognize their list membership. If this is true, then it should follow that on a recognition test, which does not require recall of items, of course, there would be no differences in performance on lists varying in interitem relatedness.

Table IV
Recognition Scores for 15-Item Lists
Varying in Interitem Associative Strength

List	Percent correct
High frequency	87.33
Low frequency	82.91
Zero frequency	86.40

I have performed one experiment in which six 15-item lists were used that varied from high to low in their indices of interitem associative strength. A recognition test was used as the retention test and contained 15 distractor items randomly mixed with the list items. The distractors were chosen from high-frequency responses given in forced recall for these lists in other experiments by other subjects. The results are shown in Table IV. There is no major effect of associative strength on recognition performance, suggesting that when items do not have to be produced, recognition of list membership is independent of associative strength. There is, however, a hint of poorer performance on the low-frequency lists than on the other lists. Although the variation is too small to be significant, it is suggestive that implicit-associative responses, occurring during list presentation, may have included distractor items. Since some high-frequency associates of the list names were distractor items for these low-frequency lists, their occurrence during list presentation is indeed plausible. Howard Walker, in an unpublished experiment, in a parallel way has found that distractor items belonging to the same categories as the correct items are more often chosen on a recognition test than noncategorized distractor items, though the effect was a small one.

Given the boundary conditions I indicated earlier in this paper, I would characterize free recall as follows: The subject, hearing or reading the list items, retains a core of items, the number being dependent on his memory span and the rate of item presentation, and the specific items tend to be those at the beginning and at the end of the list, rather than those in the middle of the list. I have not presented it here, but there is good evidence for the effects of rate of presentation on item recall scores in free recall as well as for serial position effects (Murdock, 1962). Reproducing the items he remembers, which for a given subject may be constant across lists, the subject will be stimulated, in the case of items that are interrelated, to think of other items that are not included in the core of retained items. This thinking of related items is the generation process. Having generated items, the subject will include them in his recall if he recognizes them as list members but not otherwise. College student subjects tend to be conservative; they will record items only if they have reasonable confidence that they were on the list.

IV. Implications for Psychopathology of Memory

It is probably appropriate, though perhaps gratuitous, for me to say something concerning the psychopathology of memory from the standpoint of the situation with which I have worked and the tentative model I have outlined of the processes involved in memory as reflected in this situation. There are a number of points at which the integrity of the processes I have suggested could be affected. One, of course, is the memory span and whatever perceptual registration and attentional processes are involved in it. In the broad area of pathological memory, are the factors of rate of presentation and serial position involved in the same parametric ways as they seem to be in normal memory?

I would anticipate that the first locus of pathology would lie in the memory span, that is, the size of the core of actually retained list members. But if generation and recognition are as important in list reproduction as I have postulated here, then we should see further evidence in the factors involved in them. Are categorical relations among items seen differently from the way they are in normals? What of interitem associations? What are the structures of associative relations in pathological memory? Are they as orderly and as abstract as they are in normals? Are they as free from the influence of external, situational cues as they seem to be in normals?

What criteria determine, in pathological memory, the reproduction of an item? Are they as stringent as they seem to be in normals and as related to actual list membership?

These are some of the questions which, it seems to me, are possible avenues of inquiry that may help to improve our understanding of memory, pathological and normal. And I am sure that there are many pathologies of memory, depending on a host of conditions.

I am led by this last remark to add that in generalizing about memory we must be concerned not only with the subject variable but also with task variables. I have spoken here about free recall, and in this situation interitem similarity—such as that based on conceptual and associational relations—augments performance. But in other verbal learning situations, such as verbal discrimination, paired-associate learning, and serial learning, the effects of similarity may be detrimental to performance rather than facilitative. Task and situational analyses are as integral to our study of memory as are subject variables. I am dubious, in other words, about the potential value of simple, sovereign, one-process conceptions of memory that are applicable, as some theorists seem to suggest, to memory phenomena no matter what the situation in which they occur.

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THE ORGANIZATION OF LEXICAL MEMORY: Are Word Associations Sufficient?[1]

George A. Miller

The aspect of memory that I want to discuss is rather special and perhaps, relative to the central topic of this book, of marginal interest. It concerns some relations between memory and language. For several years I have been curious to know how our ability to speak a language affects our ability to memorize and recall messages in that language or, if you prefer, how our very long-term linguistic habits support our shorter-term verbal learning. Another way to ask the question is this: How is human memory organized in the service of language?

Studies of verbal learning and memory in normal people imply that ease of learning and accuracy of recall depend in large measure on the extent to which previous learning can be brought to bear, that is, on the extent to which a new task already has been mastered before we encounter it. For example, a meaningful sentence is relatively easy to recall because it conforms to well-established linguistic practices. A list of nonsense syllables is hard to remember because our speech patterns are of little use; we have to learn more in order to master the nonsense task. One way to characterize this transfer of training is to say that our knowledge of English enables us to recode grammatical

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strings of phonemes into familiar sequential units, and the recoded units are easier to learn and remember.

I do not know what light the observation of memory pathologies or language impairments might shed on this general account, but I believe that the transfer hypothesis is well supported for normal people both by experimental evidence and common sense, so I will not attempt to defend it here. It provides a background for the following speculations, which I hope may be suggestive to clinical investigators.

The transfer hypothesis becomes a bit vague, and its adherents begin to disagree, when one asks precisely what it is in our store of well-established linguistic habits that is transferable to new situations. At one time I believed that familiarity with the sequential aspects of sentences made all the difference. I thought that sentences must be composed of familiar sequences of verbal responses, of sequences of words heard and produced frequently in contiguity. Prior experience would strengthen particular sequences of words, which then could be transferred to sentences, but not to nonsense strings. Now I no longer believe that habitual sequences of words can be the whole story. For one thing, the variety of grammatically admissible sequences of words is so great that we could not possibly have heard all of them previously, much less developed them into habits. For another, every user of the language can recognize admissible sequences even when they are completely novel. Moreover, sentences by and large are meaningful; our ability to comprehend their meanings also has something to do with speed of learning and ease of recall. In most situations we are more likely to remember the "gist" of what we heard than to remember particular sequences of words. Mere recoding is not enough; the recoded version must be meaningful. How this subjective process of understanding should be described is no simple question.

How, then, should we characterize transfer from knowing how to speak to remembering what was said? One familiar approach says that this transfer is mediated by preestablished "associations" among (content) words in the sentence. The usual assumption seems to be that the process of understanding a sentence is accomplished when associations are established among its component parts; the difficulty of the learning task is determined by how many of the associations are new and how many are already familiar by virtue of one's knowing the language. The greater ease of recall for meaningful sentences is attributed to preestablished associations that can be used to reconstruct parts that otherwise might be forgotten.

In order to study such hypotheses, some psychologists have devoted considerable attention to word association tests – "Give me the first word you think of when I say *X*"—since that procedure seems to provide a convenient way to explore the intricate net of relations that exists among words. It is obvious, however, even from data obtained by that simple method, that there are many different kinds of relations among words. The person who responds *able* to *table* has one relation in mind, the person who responds *chair* has another, and the person who responds *eat* differs from both. Attempts have been made to classify such responses; the results have never been very satisfactory, however, because there is considerable ambiguity about the subject's intentions inherent in the method. In any case, many association theorists would like to simplify their basic assumptions by deriving all these apparently diverse relations from a single principle of learning, namely, association by contiguity, so the differences among them may have been somewhat underplayed in psychological writings.

One result from word association tests, however, was the discovery that association is often asymmetric. For example, if *black* is the stimulus, *white* will be the associated response more often than *black* will be the response to *white*. One explanation that has been offered for this directional asymmetry is that the phrase "black and white" is more frequent (it is alleged) than "white and black" in everyday speech. According to this account, word associations draw strength from two rather different sources, which have been dubbed "paradigmatic" and "syntagmatic."

Two words are considered paradigmatically similar to the extent that they are substitutable into identical contexts and syntagmatically similar to the extent that they follow one another. *Cat* and *dog* are paradigmatically associated, since they have similar privileges of occurrence, for example, they can be modified by many of the same adjectives or can serve as subjects for many of the same verbs. There is little reason to expect that *cat* would evoke *dog* more often than *dog* evokes *cat*—unless, of course, "cat and dog" is more frequent in everyday usage than is "dog and cat." It is the syntagmatic component, presumably, that contributes a directional difference. For example, the stimulus *doodle* almost never evokes the response *Yankee*, but *Yankee* frequently evokes *doodle*, for syntagmatic reasons.[2]

Thus a directional difference in strength of association, it is claimed, can show what kind of association is involved; of course, we have no assurance that this is the only source of directionality. I would like to focus on this asymmetry not because it is an important phenomenon in its own right but because it is sufficiently simple that we might be able to account for it.

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[2] Young children give relatively more syntagmatic associations than adults; the "paradigmatic shift" is a part of growing up. It would be interesting to know whether pathological effects on language and/or memory would affect this adult preference for paradigmatic responses.

Let me discuss these phenomena in the context of a particular kind of relation between nouns, a relation that has been of interest at least since the time of Aristotle, namely, relations of class inclusion. Knowledge of these relations is in some sense obligatory; anyone capable of understanding sentences must have learned these inclusion relations. For example, in order to understand the sentence *Lassie is a collie* I must know the meaning of *collie*; it is a part of the meaning of *collie* that collies are dogs and that dogs are animals and that animals are physical objects; if I do not know all that I simply do not know the meaning of *collie*. I take it as axiomatic that such inclusion relations form a part of our lexical memory. And I also assume that knowledge of these relations is manifested when subordinate or superordinate responses occur in the word association test.

The two questions that I have been pondering are simply these: (1) How does our knowledge of these relations affect new learning and are these the previously learned associations that facilitate the learning of new verbal patterns? (2) What directional effects in association are found as a consequence of these obligatory inclusion relations?

As to where we should search for answers, I have been able to think of six distinct hypotheses that, on *a priori* grounds, seem to have plausibility. I would like to take a few minutes to characterize them in very general terms. I should say at the outset that in my opinion these hypotheses are independent but not mutually exclusive; they may be merely different aspects of a very complex process or even different strategies a person can use in order to simplify new learning tasks. (It will be convenient to give them names, but the names used here are merely my own suggestions.)

I. Sequential Frequency Hypothesis

This is essentially the hypothesis I have just described, namely, that strength of association is determined exclusively by frequency of cooccurrence. To explain transfer to new learning, the argument is simply that the sequential associations that have been vastly overlearned in ordinary usage have already acquired strength and so will not need to be strengthened further in the course of verbal learning experiments. The sequential frequency hypothesis makes no *a priori* prediction about directionality of association; if the sequence of words *collie* *dog* occurs more frequently in ordinary speech than does the sequence *dog collie*, then that direction should dominate in the results of association tests. Why one or the other sequence should be more frequent in ordinary usage is left unexplained. (I call this the sequential frequency hypothesis, rather than the syntagmatic hypothesis, because the strength of syntagmatic associations—where syntax is conceived more broadly—need not be determined entirely by frequencies of co-occurrence.)

II. Shared-Name Hypothesis

The idea here is that no direct associations are learned among the words *collie*, *dog*, and *animal* but that indirect associations among these words are mediated by the fact that some objects share all three names, some share only the last two, and some have only the last one. In the simplest version, a child learns each of these names separately for the same canine referent, and only later (if ever) becomes aware of associative relations among the names. An aphasic patient who could assert, for example, that a certain object was a *dog* but be simultaneously uncertain whether or not it was an *animal* would provide strong evidence for this kind of semantic independence.

III. Image Hypothesis

Suppose that a word evokes a mental image. Suppose, for example, that the noun *animal* evokes an image of an animal. At least since the time of Bishop Berkeley we have recognized that an image must be particular; an image of an animal must be an image of some particular animal. Therefore, if the particular animal image happens to be a dog, then *animal* has caused us to think of a dog. But a dog image also must be particular. Suppose it is a collie; then *animal* has caused us to think of *collie*. But even *collie* is the name of a class, so we must have had the image of some particular individual collie, for example, *Lassie*. Unlike the shared-name hypothesis, an image hypothesis seems to go best from superordinate to subordinate. Presumably, new learning will be facilitated to the extent that it evokes strong imagery, and the image evoked by a sentence generally will be more unitary than a sequence of images evoked by a random string of words. The memorability of images long has been recognized; it provides the basis for most of the standard mnemonic devices.

There would seem to be both a voluntary and an involuntary version of the image hypothesis. According to the voluntary version, a person can decide in advance that he will adopt imagery as his strategy for coping with the demands of an association test. Whenever possible, therefore, he tries to form an image of the referent of the stimulus word and finds a response word by examining this image with his inner eye. The involuntary version would hold that the person had no choice; he must always respond in terms of some kind of imagery.

IV. Branching Hypothesis

If we take a more structural approach, we recognize that the terms *collie*, *dog*, and *animal* can be located on one branch of an extensive taxonomic tree that also includes *bulldog*, *terrier*, *retriever*, and so forth and *cat*, *chicken*, *elephant*, and so forth, and even plants as well as animals. If we assume that everyone who knows English has learned some such branching structure, then at least part of understanding the word would be a matter of knowing its location in the taxonomy. In particular, the direction of association would seem to flow better from subordinate to superordinate, from many stimuli to one response, rather than vice versa. That is to say, from every twig we can find our way back to the same trunk of the tree, but when we must move from trunk to twig we face an overwhelming variety of paths.

V. Semantic Marker Hypothesis

According to one view of these matters, the structure of a lexicon can be best characterized by semantic markers. Each lexical entry can be thought of as including, in part, a list of abstract semantic features that serve to distinguish its meaning from the meanings of all other (or most other) entries. Although it is a mnemonic convenience to assign familiar names to these markers, they are not themselves words but are abstract concepts or properties; they may or may not be represented in English by single words. In the simplest version of what a marker could be, we might have such abstract features as "Proper?" and "Count?." For example, English nouns are generally divided into proper and common; this categorization can be characterized by a marker—call it Proper?—that can have one of two values: Proper—, or unmarked, for common nouns, and Proper+, or marked, for proper nouns. Common nouns might then be further categorized as count nouns or mass nouns: such mass nouns as *virtue*, *sugar*, and *weather* would take the value Count−, and such nouns as *dog*, *foot*, and *school* would be marked Count+. In such a scheme, *animal* will have (at least) the following features: Proper−, Count+, Living+, Plant−. Then *dog* will have all the marks that *animal* has, plus something more, for example, Nonhuman+, Canine+. *Collie* will have all of dog's marks, plus something more to distinguish it from all other breeds of dogs.

Note that *collie*, *dog*, and *animal* have several markers in common. A natural psychological hypothesis would be that responses on word association tests tend to be words having many of the same markers or, if I may put a mentalistic phrase to it, words that presuppose the same ideas will tend to be associated.

When words are strung together grammatically to form sentences, a person who knows the language will know the rules for combining these semantic markers to construct an interpretation of its meaning. This synthesis cannot be performed for a nonsense string of words. An ungrammatical string of words is more difficult to remember, therefore, because in a real sense it is much longer than a sentence; its elements do not meld into fewer, larger, meaningful "chunks." According to this hypothesis, then, transfer of training to new sentences is mediated by the language user's knowledge of rules for combining word meanings into phrase and sentence meanings. A person who through some unfortunate accident had lost his mastery of these rules might know names but not be able to interpret sentences; the transfer from knowing how to speak to recalling what was said presumably would be lost in such patients. A complementary picture would be expected if a person retained the rules for combination but lost some of the semantic markers.

According to this semantic marker hypothesis, a directional effect in word association data presumably would be expected to go from subordinate to superordinate, on the principle that it is easier to delete than to supplement. Given *dog* as a stimulus, it is simpler to drop a few markers, thus arriving at *animal*, than to add the markers necessary to designate a particular instance of dog.

In the case of *collie*, *dog*, and *animal*, the semantic markers define a graph that is much like a taxonomic tree, so it might seem that a semantic marker hypothesis and a branching hypothesis are indistinguishable. In fact, however, there are important differences. For one thing, the branching hypothesis (as I have presented it here) has words, rather than abstract concepts, at the nodes of the tree. For another thing, semantic features need not result in a branching structure at all; kinship terms, for example, seem to be much more efficiently classified (nearly every term can be marked for nearly every feature) than are animal names. A suburb of the lexicon could be well organized by semantic markers without being representable by a tree graph.

VI. Predicate Hypothesis

Finally, we might base a hypothesis on the fact that associated words generally can enter into a simple predicate relation: *A collie is a dog* and *a dog is an animal*. One way to state this hypothesis is to say that *is a* is a function that takes two arguments; *X is a Y* can be thought of as *F(X,Y)*, where *F* is the *is* of inclusion and *X* and *Y* are the arguments—for example, *collie* and *dog*. In associative terms, *is a* is the link that associates these words. If the lexicon has been learned in such a way as to support the formation of simple sentences of this kind, then the basis for transfer to sentences becomes obvious, if not tautological. (Indeed, the sentence frames *A S is a R* and *A R is a S* provide the most convenient tests for identifying superordinate and subordinate responses.) Moreover, association should run best in the direction from subordinate to superordinate, since the reverse direction of predication (for example, *A dog is a collie*) is inadmissible (except, perhaps, as a symptom of verbal confusion).

A predicate hypothesis, like an image hypothesis, can be used in two rather different ways to interpret data from word association tests. In one version, a person voluntarily can decide in advance that he will use predication as the source of his response. A subject using this strategy would deliberately select *is a* even before the stimulus word was presented. As soon as he heard, say, *dog*, he would have *A dog is a*. Finding a response, therefore, would be comparable to completing a sentence. The second, or involuntary, version would be that superordinate associations, inclusion relations, and *is a* predicates all represent one and the same psychological process, which manifests itself differently in associative, semantic, and syntactic contexts. The subject has no option to use or avoid predicate constructions; these syntactic relations are an integral and inescapable feature of the underlying psychological process that occurs when the stimulus word *dog* is perceived.[3]

These, then, are six alternative hypotheses. We can think of our lexical memory as organized into (1) a storehouse of responses – words and clichés – loosely connected by a web of generalization gradients determined by ecological contiguity; (2) a list of stimulus-response pairs – referent-name associations – basically independent but related by virtue of multiple naming; (3) a picture gallery of images, along with associated names, organized by perceptual similarity; (4) a taxonomic tree with a node for every word and every word in its node; (5) a meticulously cross-referenced catalog of abstract semantic concepts, where certain packages of concepts are represented by words; and (6) part of a device for generating sentences. Each notion has some virtues, and it is important to remember that they are not mutually exclusive. As for directionality, according to four of the hypotheses I have described, associations should be stronger going from subordinate to superordinate (from *collie* to *dog* to *animal*) than vice versa. The sequential frequency hypothesis is noncommittal. Only an image hypothesis might suggest the opposite direction. On a democratic basis, therefore, the direction from subordinate to superordinate should carry the day.

Fortunately, however, we can do better than vote. Results from word association tests show that a superordinate response will be much stronger (more frequent and probably faster) than a subordinate response. For example, when *dog* is the stimulus word,[4] then such responses as *canine, mammal, animal, living thing,* and *object* would be classed as superordinate, and such responses as *collie, St. Bernard,*

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[3] Some care is needed to keep clearly in mind the differences between a predicate hypothesis and a sequential frequency hypothesis. Unlike the argument for a sequential frequency hypothesis, no appeal is made to frequency of co-occurrence; the frequency of this particular kind of predication may be important, but not the joint frequencies for the nouns that enter into it. Nor are the associated words immediately contiguous; a sequential frequency hypothesis might be hard pressed to explain why the most common response to *dog* is not *is*, but the predicate hypothesis would not be concerned with such surface details. Nor is the semantic relation among the words ignored; only words bearing particular semantic relations to one another can serve as arguments for the inclusion function. The sequential frequency hypothesis applies to any common sequence of words; the predicate hypothesis applies only to nouns (or noun phrases) related by the predicate *is a*.

[4] Available data were averaged over all instances of subordinate and superordinate responses to many different stimuli. I use *collie, dog,* and *animal* merely as examples, in order to make the discussion somewhat less abstract.

German shepherd, and *poodle* would be classed as subordinate. When a lot of different stimulus words are presented to a lot of people and the number of responses in each of these two classes is totaled up, it is generally found that there were more superordinate than subordinate responses. For example, Woodrow and Lowell (1916), who classified the data of Kent and Rosanoff (1910), reported that 7.6% of all the responses given by adult subjects were superordinate and only 1.6% were subordinate. (The corresponding figures in Woodrow and Lowell's own data for children were 3.7 and 2.1%.) If the list of nouns selected by Kent and Rosanoff is representative of the language, then the *dog-animal* direction of association is stronger than the *dog-collie* direction.

In order to check this finding, the responses obtained from college students by Russell and Jenkins (1954) were classified by two judges as superordinate or subordinate, where the test was substitutability into the following frames:

Superordinate = *A* (stimulus word) *is a kind of* (response word)
Subordinate = A (response word) *is a kind of* (stimulus word)

The classification was done only for certain stimulus words, namely, those 52 nouns on the Kent-Rosanoff list that can be commonly used as count nouns; it was felt that subjectivity of classification could be kept to a minimum in these cases. The results, which are summarized in Table I, confirm the directional effect reported by Woodrow and Lowell: 9.6% of the responses to these nouns were superordinate and only 4.5% were subordinate.

Table I
Tabulation of Superordinate, Subordinate, Part-Whole, and Whole-Part Responses to 52 Nouns in Russell and Jenkins (1954) Data for 1008 College Students

	Superordinate	Subordinate	Part-Whole	Whole-Part	All Others
Response types	95	253	126	227	4516
Response tokens	5040	2350	3984	5031	36,011

If we accept such evidence as relevant and appropriate, then it would seem to eliminate an image hypothesis as an explanation for directionality. An image hypothesis is more appealing in a slightly different context, however: Consider next the relations among three different words, namely, *toe*, *foot*, and *leg*. Here, of course, we have shifted from inclusion relations to part-whole relations, but I think they form an equally obligatory part of the lexical information every

English-speaking person must know. If you do not know that a toe is part of a foot and a foot is part of a leg, then you simply do not understand the meaning of *toe*.

An image hypothesis might seem plausible in this context, since an image of the whole presumably includes images of its parts. Thus, the image hypothesis would suggest that association should move most easily in the direction from *leg* to *foot* to *toe*.[5] And, if we can trust data from word association tests to settle the matter, this indeed seems to be the case. Woodrow and Lowell report twice as many whole-part associations (2.1%) as part-whole (1.1%) by adults. (Comparable figures for children are 3.6 and 0.4%, respectively.) In the tabulation of the Russell-Jenkins data, it was found (see Table I) that 9.6% of the responses to the 52 count nouns could be fitted into the frame A (response word) *is a part of a* (stimulus word) whereas only 7.6% could be substituted into the frame A (stimulus word) *is a part of a* (response word), thus confirming the directional effect found by Woodrow and Lowell, but less emphatically.

One who favors a voluntaristic account could (and probably would) argue that whole-part and subordinate associations are mediated by imagery whereas part-whole and superordinate associations are mediated by something else. For example, a person who had adopted a predicate strategy would respond to *leg* with *limb*, but if he had adopted an image strategy, he would respond *foot*. Such an explanation is not impossible, of course, but there might be other alternatives. Consider again the six hypotheses, but now with the part-whole directionality in mind:

1. Sequential frequency hypothesis. Presumably an advocate of this hypothesis would expect to find *foot toe* occurring more often than *foot leg* in ordinary usage. Since neither sequence occurs in normal usage, the hypothesis again provides no explanation of the directional results.
2. Shared-name hypothesis. Since there is no body part that shares the names *toe*, *foot*, and *leg*, this hypothesis cannot be generalized beyond the inclusion relation. It predicts nothing for part-whole associations (unless synecdoche is invoked, but then the predication seems to go in the wrong direction).
3. Image hypothesis. This hypothesis might account for the data on direction of association, but we have already discredited it on evidence from superordinate associations.

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[5] Again, these particular words are chosen only for illustrative purposes.

4. Branching hypothesis. A tree graph characterizing the structure of associations among names of body parts resembles a taxonomic tree in the matter of branching, so a branching hypothesis can be generalized from inclusion to part-whole relations. It would again predict that twig to trunk (part to whole) would be stronger than vice versa, which eliminates the branching hypothesis as an explanation of directionality.

5. Semantic marker hypothesis. I am somewhat at a loss to know how to evaluate this hypothesis, since I am uncertain how markers for wholes and parts should differ. I assume, however, that the meaning of such count nouns as *foot* would have to include markers that indicated explicitly either (a) what they were part of, (b) what parts they had or (c) both. In many respects these markers would resemble associations, but they would not be neutral, anonymous associations; they would be part-whole associations, and have a unique character of their own relating some nouns, but not others, in this particular fashion. If we take alternative (a), we predict that the direction of association should favor part-whole over whole-part, which contradicts the available data. Alternative (b) would predict the right direction. Alternative (c) would have to be supplemented by further assumptions in order to say anything about direction. The most we can conclude, therefore, is that a semantic marker hypothesis is not necessarily inconsistent with the facts of directionality of association.

6. Predicate hypothesis. The predicate relation that supports part-whole associations is *has a*: *A leg has a foot*; *A foot has a toe.* Note that this subject-predicate relation orders the words from whole to part, which is in accord with the empirical observations on direction of association.

Of the six hypotheses, therefore, the predicate hypothesis is the only one that accounts simply for the directionality of word associations for both inclusion and part-whole associations. Thus, if we are persuaded by this kind of evidence, we are left with the notion that inclusion and part-whole associations show directionality on word association tests because our lexical memories are organized so as to support subject-predicate constructions of the form *X is a Y* and *X has a Z*. This conclusion supports the general opinion that directional preferences in association can be attributed to syntagmatic factors.

Is there a circularity here? Review the following argument: Sentences are easier to remember than nonsense because we can understand their meanings. Meanings (according to traditional belief) can be characterized in terms of lexical associations. Lexical associations can be measured by word association tests. The results of word association tests can be accounted for in terms of particular types of sentences we can form with the words. Therefore, sentences are easy to remember because we understand sentences, which seems to bring us back to where we started.

However, it is not difficult to break into this circle. Circularity develops only when we try to explain our understanding of sentences in terms of some prior, independent associations among words. If we turn the argument around, all is once again reasonable: We first learn how to produce and understand sentences, and that explains why we give the word associations we do. The simple stimulus-response bonds between words that seem to be demonstrated in word association tests are not the psychological atoms out of which all speech is built. Rather, they are a consequence of making people use their linguistic competence in an unusual, not to say aberrant, test situation. If we could first characterize how a person uses sentences, then we might be able (if anyone still cared) to explain why he behaves as he does on tests of word associations. Efforts to move in the opposite direction, however, have had little success.

For students of verbal memory, both in the clinic and the laboratory, I offer the following homily: Speech is not the passive victim of whatever lexical associations we happen to know; lexical memory must be organized in the service of speech. When we talk we generally designate some topic and then ascribe some property to it. This basic subject-predicate union characterizes the propositional content of all illocutionary acts. It would seem to imply, for example, that lexical memory should have at least two different kinds of entries, one suitable for identifying topics of sentences and the other suitable for serving as predicates. If a predicate hypothesis were to be taken seriously, we might even assume that all predicates *impose* organization on the names, just as *is a* imposes an inclusion relation and *has a* imposes a part-whole relation. This is not the time or place to speculate about the cognitive machinery needed for processing sentences. I merely wish to suggest that when we do have such a theory we will not find that word associations per se play any important explanatory role in it but, on the contrary, patterns of word association should fall out as a special and rather artificial by-product of machinery designed for a much more important purpose.

In closing I should like to say that I personally believe that some combination of the semantic marker and the predicate hypothesis is required to account for our linguistic abilities. A semantic marker system must be supplemented by rules for combining markers in grammatical phrases. The *is a* and *has a* relations discussed here may be simply two such rules, in which case we probably should call them together the grammatical function hypothesis, where predication would be only one of several functions for which we know the rules.

This way of thinking about the problem makes it far more complicated than most psychologists would feel is necessary in order to account for the facts of verbal memory. Certainly I have balanced here a very extensive theoretical analysis on a pinpoint of empirical fact about transfer and directionality of association. Once the hypotheses are clearly formulated, however, it is not difficult to think of new procedures for testing them more directly. The real justification for such a complicated theory is, I believe, that it is in reasonably good agreement with the kind of information that linguists say a language user must have anyway. And I suspect that explanations of at least this degree of complexity eventually will be required in order to account for the complex relations among language, lexical memory, and learning ability in neuropathological states.

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THE PRECONSCIOUS FACTORS IN THE PROCESS OF REMEMBERING[1]

Lawrence S. Kubie

My working hypotheses are (1) that the study of remembering is inseparable from the study of forgetting, (2) that both together are inseparable from the study of all thinking processes, and finally (3) that no further studies of the conscious symbolic components of remembering, forgetting, or thinking can be meaningful unless based upon a far more difficult but even more essential study of the underlying preconscious stream.

I. The Dependence of Selective Remembering on Selective Forgetting

An engineer pointed out that there can be no communication apart from concurrent selections that are based on massive concurrent rejections. The same is true for remembering. In fact, every process of retrieval makes available only a sample out of the total product of a complex process, which involves even more selective exclusion and "forgetting" than remembering. Conscious recall is the symbolic representation of an even smaller sample out of the first sample and de-

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pends upon more selective exclusion or "forgetting" than "remembering." The final yield is only a small weighted fragment of the whole. Clearly the two processes of remembering and forgetting cannot be studied apart from each other, nor apart from other contributory processes, such as massive input, selective perception, and data processing and its symbolic representation. This is why the word "memory" is a misleading term. "Memories" are items retrieved and coded. "Remembering" is the process. Memory is a mere abstraction. Furthermore, the underlying process of remembering is not carried on consciously but preconsciously through concurrent preconscious selection and rejection or "forgetting." The conscious product is the symbolic coding of a small weighted sample of this. Each individual act of remembering depends upon a mixture of all these processes. Consequently the process of remembering cannot be studied as a unit or as a thing apart. Its several ingredients are what must be correlated with anatomical, pathological, physiological, biochemical, affective, and general experiential or personality variables, since variations in the end product of the process of remembering are produced by variations in any or all of the contributory steps. If we allow ourselves to lump together all of the quantitative and qualitative differences among the ingredients in the processes of remembering, subsuming them under such a term as the "psychopathology of memory" (as though the abstraction called memory were a unit), we will end by trying vainly to match precise somatic variables (or less precisely observed and recorded psychological variables) with a nonexistent abstraction. This is the familiar fallacy that occurs whenever abstractions are reified. Neuroanatomists, neuropathologists, neurophysiologists, experimental and clinical psychologists, psychiatrists, or psychoanalysts all have been misled when they have approached the problem in this way.

These considerations lead me to make one further general comment: Nouns get us into trouble. This is not the first time that I have suggested that the student of psychophysiology and/or of psychology is always on safer ground when he uses adjectives than when he is misled by nouns. Perhaps the only way in which to be sure of avoiding the fallacious reification of abstractions is to eschew the use of nouns altogether. However, if we cannot do without them entirely, then we must learn to use them with special care.

In varying degrees every noun, even so simple a noun as the word "chair," is abstracted from a generalization made swiftly and preconsciously from multiple observations of similar, overlapping, but far from identical bits of informative experiences. Thus, when we bring together under one heading a dinner chair, a high chair, an electric chair, a musical chair, and a chairman's chair, we create an abstraction from multiple experiences. It has some attributes in common but more differences. It has no existence except in our heads and no qualitative or quantitative variables of its own. What we do with this simple abstraction, we do more flagrantly to such an abstraction as memory. Having given it a name, we then tend to attribute not only qualitative and quantitative variations to this named abstraction but also independent powers. We speak of memory as good or bad, better or worse, strong or weak, intact or full of islands. Such words are useful only as long as we do not allow ourselves to forget for a moment that they are descriptive metaphors. They do not explain; they only challenge us to ascertain to what ingredients in the total process they are relevant, how these apparent variations are produced, and what their results are. These are the questions that must be answered if our investigations are not to remain cloudy.[2]

It may help us to understand the importance of these observations if we consider how a failure to formulate issues clearly has muddled the approach to other psychophysiological processes. I have in mind the deliberations of the Laurentian conference on consciousness in 1953 (Kubie, 1954). An attempt was made there to persuade the meeting of the importance of this issue but failed in spite of the fact that it had the support of William James' classic paper, "Does Consciousness Exist?" (1912). The conferees nevertheless failed to take any notice of the distinction (Kubie, 1954), that is, between consciousness as a reified abstraction that cannot be studied and consciousness as a process that can be investigated. It was pointed out that the essence of consciousness as a process is a selective sampling based upon rejecting and that this sampling is of an underlying stream of continuous central activity that is itself preconscious [that is, imageless (Humphrey, 1951)] and that consciousness as a process enters the stream of psychological events only when these fragmentary and weighted samples are represented by symbolic images. Thus, like the process of remembering, the consciousness process is a condensation of many intermediate steps, the variations in each of which must be studied if we are to illuminate the consciousness process as a whole. Conscious and preconscious input together supply the bits of information for the subsequent preconscious processes of rejecting, which are prerequisite to all selecting and to the subsequent ordering, summating, coding, sampling, and symbolic representation of the underlying stream.

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[2] The fallacies that result from the reifying of abstractions lead directly to anthropomorphic fallacies. In fact all psychologies, including psychoanalysis, provide examples of anthropomorphic distortions of reified abstractions. Therefore, I would emphasize again the fact that when neuroanatomy, neuropathology, neurobiochemistry, and neurophysiology borrow psychology's fallacious abstractions and match them against their own more precise anatomical, pathological, and physiological data, this lopsided process merely multiplies the fallacies in their joint conclusions.

For the purposes of this symposium on remembering and forgetting I wish that I could have the support of an authority comparable to that furnished for my argument by William James (1912) at the Laurentian conference. I can conjure up an imaginary paper, perhaps by the late David Rapaport, which he might have called "Does Memory Exist?"

This description of the consciousness process is identical with the description of the process of remembering. In each, the symbol represents only a weighted, coded sample of an underlying preconscious stream. In the consciousness process the emphasis is on the use of the process for perceiving, ruminating, communicating, and reality testing. In the process of remembering the emphasis is on the retrieval of past items out of the same matrix. In both, the outcome depends first upon the balance among concurrent processes of sorting, rejecting, and selective sampling and then upon the balance between items that are represented by symbols where the relationship between the symbol to its referent roots is undistorted and intact and, on the other hand, other items in which this relationship is distorted or severed. Here of course is where the process called "repression" and the process called "forgetting" converge.

The processes of symbolic coding and of symbolic representation of fragmentary and weighted samples of the continuous stream of preconscious processing constitute only the final steps both in the consciousness process and in the process of remembering. Future investigations of the two closely related processes cannot be limited as they have been in the past to the study only of the vicissitudes of the final step but must turn their attention to the vicissitudes of the underlying preconscious stream itself.

As we study the processes of remembering or, for that matter, any other psychological processes, it might help us to communicate more clearly over our interdisciplinary gaps if we keep in mind a few other general principles. I lay major emphasis on a developmental approach: The study of the development of each ingredient from its earliest steps in infancy to its fully matured expression. (This approach is implicit in the brilliant contribution Jerome Bruner has made to this book.) The developmental approach would begin with the early acquisition of preconscious functions through processes of automatic learning and identification and also through conditioning

(Kubie, 1959). It then would move to the ontogeny of symbolic processes. In somatic medicine as in psychological medicine the understanding of normal ontogeny furnishes an essential baseline for understanding how development can be disturbed or blocked under the influences of structural, anatomical, genetic, neurophysiological, and neurobiochemical variables and also under the variable somatic influence of human experience (viz., the psychosomatic level) and how each of these can initiate linked chains of reverberating consequences. Such developmental studies can never be made directly on the whole end product as such. Instead they must be made on the various ingredients in the process. None of this is easy; but none is impossible. Such a developmental approach to pathology is not peculiar to psychological studies. We do not study any terminal state of health or illness as a unit: We break it down into the processes by which health is achieved and maintained and into the several ingredients in the processes of illness and their intermediate steps. Therefore, what I am urging is only that in future studies of the ontogeny of processes of remembering we be guided by principles that govern all studies of human biology.[3]

My second emphasis is on the importance of starting any study of the processes of remembering by asking *what* it is that is remembered or *not* remembered. This is never an isolated item, because we never register or process or retrieve an isolated item of experience. Whether the input arises in the soma or in the outside world, any perceptual event, even the most elementary bit of information, becomes the center of a network of related associative linkages that develop during the subsequent central processing. The majority of these links are subliminal or, as I prefer to say, "preconscious" because of the preparatory significance of the process. Elsewhere (Kubie, 1934, 1942, 1959) I have pointed out that this network arises in part out of contiguities in time and space by a process related to conditioning. It is further shaped by similarities and dissimilarities, by comparisons and contrasts, by true and false analogies, by swift appraisals of size and weight (Petrie, 1967), and so on. All of these contribute associative pathways to the constellation that is the unitary datum to be retrieved and tagged. Any isolated fact, for example, a chair or stick or pain or person, stand out as the conscious symbolic representative of the whole, the rest comprising part of what the Würtzburg School

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[3] Strangely enough, those who study the somatic variables take this principle for granted when they study somatic disease but tend to brush it aside as an unnecessary complication when they study experiential and psychological levels of organization and disorganization.

(Humphrey, 1951) called "imageless thought." But it is the total constellation that we retrieve as we use some verbal or visual hieroglyphic to represent the rest and at the same time to mask it. The small sample of all this that we call to mind consciously by means of a symbolic representative is at most a stand-in for the total net. A sophisticated study of the processes of remembering and forgetting must include it all. In the light of such considerations as these I am further troubled by the title of the symposium with its misleading implication that thinking and remembering are different processes and also that remembering and forgetting are separate processes, when in fact all are one.

One of the most important capacities of the human thinking machine is its capacity to combine opposites, to bind space and time, to bring together the near and remote, to link all into one integrated and continuous unit. This also implies that all thinking, hoping, planning, and anticipating necessitates not only recalling but also selecting, rejecting, and forgetting. Consequently, we cannot study the processes of remembering as though they were wholly apart from and different from all perceptual, cognitive, and planning procedures. A clear approach to the processes of remembering should be preceded by establishing some working hypothesis concerning the nature of all of these processes, which the workers from each of the contributory fields then can agree to use.

II. The Ingredients

Let me now review some of the ingredients in this chain of psychological processes, each link of which must be sound if the process and its remembered end products are to be unimpaired:

1. Obviously any remembering must be based upon a preceding process of experiencing and registering input that leaves something to retrieve. This, however, is also a complex process with several ingredients, for example, perception with its many variables; the registration of input on a conscious symbolic level; the registration of preconscious input and also of mixed conscious and preconscious ingredients; and a subsequent overflow into both skeleto-muscular and autonomic responses, which thereupon add their own bits to the remembering of the total input to be registered and processed. These last are frequent and important and at the same time elusive ingredi-

ents in the process of remembering; they also are most difficult to study. The fact that such circular relationships are ubiquitous contributes to the complexity of the phenomena of remembering.

2. Naturally there are further variables in the preconscious processing of bits of information after input, in their ordering and coding for the process of internal summating and coded signaling, which is the precursor to a final sampling of the preconscious stream of activity for appropriate symbolic representation.

All the above are ingredients in the day-by-day process of experiencing events as they impinge upon our receptive apparatus, whether from the outer or inner world or from the surface that divides the two —in short, of exteroceptive, enteroceptive, and proprioceptive ingredients. Each of these variables plays a role in the processes of remembering.

3. The conscious and preconscious processing of data establishes the pathways of association. Initially these arise through the propinquity of the original units of input in time and/or space. The process is closely allied to the well-known mechanisms of conditioning. Without such associative pathways no bit of information, however well imprinted it might be, could be accessible to any process of recall or retrieval. It would be like a railroad station standing isolated in a field with no rails leading to or from it. Therefore the richness, abundance, range, and variety of conscious and preconscious associative links determine the accessibility to recall; and everything that varies the establishing or maintenance of associative links is of vital importance for the process of retrieval that we call remembering. Yet only rarely have we seen them studied in research in this field. The major studies of this aspect of the problem have been psychoanalytic. Surgeons, pathologists, and physiologists have overlooked it.

4. The ability to use verbal and other types of symbols in order to represent accurately that which is recalled will determine not the intactness or persistence of the imprint of experience, but its accessibility. Therefore any disorganization of symbolic functions will disturb not the existence of imprints of past data or the capacity to bring the past into the present, but the accuracy of its representation. The effective usefulness of the processes of remembering is vulnerable to such disturbances as the aphasias and the anomias. In all forms of brain damage this makes it difficult to determine whether remembering, or whether its symbolic representation and communication, is impaired.

5. In the process of remembering, reliving is yet another ingredient of special importance both in the neurotic and creative processes.

Something can occur in varying degrees that can best be described as a reexperiencing of the past almost as though it was in the present.

Even this list of complex ingredients is not all-inclusive. It emphasizes that remembering has many components and is not just one mental act but several, which are linked in serial cybernetic chains, each link of which is vulnerable to disturbances under the influence of somatic variables in the body as a whole and in the central nervous system and also by the effects of psychological events on all levels—conscious, preconscious, and unconscious—such as conflicts and the play of emotions such as joy, depression, anxiety, and rage. All of these influence imprinting, registering, establishing associative links, and preconscious processing and symbolic representation of the continuous flow of preconscious activity. Therefore, the freedom with which we can reach into the past and then use our symbolic processes to represent that which we recover is the final measure of the freedom of a complex chain of inner events from organogenic and concomitant psychogenic disturbances. There is no either-or dichotomy here.

Furthermore, unless we consider these ingredients in the process of remembering and their qualitative distortions and substitutions and the quantitative obliterations and intensifications to which each is susceptible, elementary confusions result, for instance, the confusion between the ability to attach symbolic codes to data and the ability to imprint, store, process, and retrieve data. Out of this arises such elementary fallacies as the prevalent idea that if a person cannot "recall" something he has "forgotten" it. Five minutes later, however, or an hour later, a day later, or sometimes years later the "forgotten" data may appear undistorted. This furnishes evidence as clear as any experimental design that in that instance at least nothing has been lost and that the difficulty has been in the accessibility of particular bits of information to recall (that is, their retrievability) or in the processes of symbolic representation of that which is retrieved. In this way an anomia can simulate an impairment of remembering.

We are exposed to still other obvious errors if we focus only on the abstract end product that we miscall "memory," overlooking the variables that can alter each of the ingredients singly and together. For instance, the entire process of retrieval can take place without images, that is, on an asymbolic preconscious level. Yet any communication or rumination about anything that is retrieved requires intact symbolic functions. Therefore, even when all of the components in the processes of retrieval are unimpaired, any impairment in its final symbolic representation can produce an illusion of a defect in the process of remembering. In fact any defect in symbolic representation may render the process of remembering ineffective, even when the process itself is intact.

III. Preconscious Components in the Process of Remembering and in Its Products

My hypothesis is that all cognitive processes as well as all remembering and forgetting depend upon the retrieval and symbolic tagging of a fragmentary, weighted sample of the continuous complex stream of "imageless" or "preconscious" processes (Kubie, 1958, 1967). This stream goes on constantly during life, from infancy to death, in sleeping as in waking. Yet there have been no adequate studies of its development and of its variations between sleeping and waking in health and in illness, in youth and in age, and also in brain damage. Variations in the processes of weighted symbolic sampling have been studied, but not variations in the preconscious stream itself. Until this is done (and it presents many difficulties) our investigations will continue to be limited to those variations in the sampling process that fall into the category of aphasia, alexia, agnosia, etc. That these are important is obvious; yet such studies leave the core of the process obscure.

It has been demonstrated clinically and also experimentally (Marsh & Warden, 1956) that preconscious input can evoke preconscious autonomic responses, as well as preconscious remembering, and that even nonverbal responses can be symbolic, as has been demonstrated in experimental observations with automatic drawing and writing, both under hypnosis (Marsh & Warden, 1956) and in specific tests (Kubie, 1935, 1952; Kubie and Erickson, 1938, 1941).

Special examples of this were furnished in experiments conducted during the war with radio operators from the radio control towers of a group of separate but related airfields. This was before the days of automatic recording, and the operators had to take down in shorthand the pilot's communications from the plane as he took off from the field. On one occasion there had been a burst of excited words from the pilot of a plane as it was falling, killing all on board. His speech had been too swift to be deciphered. Yet it was felt to be of special importance to ascertain the cause of this tragedy, and one clue to this might be what the pilot had said in his last moments. Radio operators from several fields thought that they could "remember" fragments of what they had heard; but none was certain of the words. For several weeks they were brought in for separate interviews and testing. All of these tests were carried out under the continuous and direct observation of a group of experienced flight engineers. A wide array of tests were made without drugs and under various drugs and also under hypnosis with and without drugs. Repeatedly at a moment of critical communication their own voices seemed to disrupt the state of communicative sleep however produced, perhaps by frightening them, thus confusing or dispelling the trace memories. Therefore it was decided to try silent automatic writing under hypnosis in an effort to retrieve more dependable data. Certain key words recurred in the automatic scrawls of each subject, and at the same time the automatic writing followed a consistent slope that the engineers identified at once with that of a falling plane. The combination of the words and the slope convinced the flight engineers that these radio operators were reproducing months after the event the excited rapid words they had not been able to unscramble when they were alert and conscious. The engineers were excited over this and more fully convinced that they had found the cause of the disaster than was this observer. At the least, however, the observation seemed to indicate that studies of the processes of remembering should not be limited to verbal samples, which give only a partial view of the total process. (These experimental observations were restricted until recently, and have not yet been published.)

Another example is of a woman who had lost her only child and subsequently had to undergo a hysterectomy. After this she developed a toxic infectious delirium. In this state her orientation in time and place and her formal "memory" were wholly disrupted. Yet in the delirium she hallucinated twins whom she went through the motions of nursing and diapering. Obviously she was carrying forward in this distorted way a complex residue from her painful recent past. Her memory processes were active, even if symbolic representation was distorted by combined organic and psychological processes. Such phenomena as these also must be included in investigations of remembering.

A medical student was walking in the labyrinth of English box hedge in the garden at Mount Vernon. Gradually he was overcome by a sense of poignant nostalgia, almost to the point of tears. There seemed to be no reason for this until he discussed the experience with an older sister, from whom he learned that English box hedge had abounded on the farm on which he had lived from the ages of 1 to 3, when his mother had died. Of this farm he had no conscious memory traces other than this deep olfactory imprint.

Marsh and Warden (1956) exposed a group of subjects to a list of words by means of a tachistoscope, each word being flashed for so short a time that the words could not be read. Only a scribble and the flash of light was discernible. The list included a few special words that were emotionally loaded. Some of the subjects, but not all, responded to these loaded words they could not read with measurable autonomic changes.

Comparable experiments made by Pötzl in Vienna many years ago led to more sophisticated experiments by Charles Fisher in New York (1956). Again by means of the tachistoscope, subjects were exposed to words and figures too briefly to identify them. Nevertheless, recognizable traces were reproduced in dreams and in free associations. Again this demonstrates that a subliminal or preconscious intake can shape subsequent symbolic responses, another fact which should be included in future studies of the processes of recall.

We also must note that the interposition of symbolic samples of the continuous preconscious stream can occur at many points. The preconscious stream itself has been given little attention by the clinician, by the experimental or clinical psychologist, or by the experimental neurophysiologist. This is due both to a lack of general appreciation of the importance of this component in the processes of thinking and/or remembering and also to the fact that we lack adequate techniques for the study of the preconscious core of all human mentation, thought, feeling, learning, or recalling. For this reason studies are lacking on variations in the preconscious core under the influence of organic or psychological variables. As I have said, only the conscious symbolic sampling has been studied. Of course studies of the processes of remembering and of reproducing past experiences must include the conscious symbolic samples of the continuous preconscious stream, because the weighted and fragmentary symbolic samples serve the important purposes of communication, rumination, reality testing, and finally memory testing. But memory *testing* is not the same as remembering (Kubie, 1966).

This leaves us with many important questions, some of which have never been raised and some touched on hastily in passing.

We must challenge ourselves to discover at what point in this long complex chain of confluent and reverberating events is it that anatomical, neurobiochemical, and neurophysiological variables exercise their influence. Related to this is the question of at what points do such experiential variables as motives and conflicts, whether conscious, preconscious, or unconscious, alter these processes and their end products.

The reduction of and interference with the processes of remembering should be illuminated by contrasting studies of anything that may seem to facilitate and enhance them, for instance, such drugs as Cylert and such processes as experimental hypermnesis under hypnosis, and the possible enhancing effects of aging on vividness, detail, and accuracy of recall of remote experiences and the feats of memory of the "idiot savant." But first we must ask whether hypermnesis is a fact or an illusion.

A special problem confronts us whenever we attempt to uncover changes in the underlying preconscious stream if at the same time the symbolic sampling devices are impaired as in all aphasic disorders. Yet this can be studied through more carefully controlled observations of free associations, the ratio of repetition to stereotype in free association (Kubie, 1960), and the use of automatic writing and drawing under hypnosis that can be translated (Kubie & Erickson, 1940; Kubie, 1952). One point can be made with confidence: We will not understand reductive influences on the processes of remembering unless we also can study and illuminate the processes of enhancement.

The influence of varying time intervals needs further study. Starting from the effect of immediate contiguity in time in the establishing of conditioning, we will have to ask precisely what is the limit of the time and space intervals that constitute immediate recall. Is this uniform for all people? Is it constant for any one individual? Does it alter with aging, a step by step or at some critical point? What in this connection is the range of variation among individuals or within an individual? Similarly, we do not know how long is "recent" nor how long is "remote" nor their constancy within one individual or their variability from one to another nor the influence of age or of the diurnal cycle nor their variations in sleeping and waking or under the influence of drugs such as marijuana, which seems to alter the awareness of the passage of time. These are basic in the processes of retrieval, in the recognition of familiarity, and in choices made among multiple alternatives.

In this connection the question comes up whether *immediate* retention is part of the process of remembering at all or is rather a reverberating echo. Can the mere passage of time alone erase a trace of a preconscious and/or of a conscious input? Since it seems as though processes initiated in the nervous system do not undergo spontaneous decrement but continue until some interfering process intervenes, is the influence of the passage of time merely a consequence of the probability that with the passage of time some interfering process is bound to intervene? Here again, is there a range of individual variations?

Conversely, does the passage of time ever intensify the traces of the initial input, perhaps through the added opportunity time provides for the elaboration first of the associative network and later of the symbolic sampling? Here again we confront the influence of the duration of input and its effect on preconscious registration, processing, and reproduction. The human potential for establishing reverberating circuits also may be a vital variable in the transition from instant recall to recent and remote memory.

Each of these items is influenced by the concurrent play of affective attitudes. Unfortunately, however, these have been studied more in the confusion of the clinic than in the precision of the laboratory. Also they have not been subjected to clear analytic investigation under hypnosis.

Further investigations must be made to clarify the variables that affect the input process, that is, how much time does it take to register preconsciously or consciously and how many organic and psychological variables influence this? How long an interval between individual bits of input is required to give each item separate identity in the input system? What is the influence on this of a concomitant preconscious bombardment? The answers to such questions as these will throw some light on the speed of preconscious processing and also on the immediacy with which the symbolic sampling can begin. (Incidentally, for those who are interested in the influence of the variable ingredients in the processes of remembering in the learning process, such studies will throw light on the extent to which so-called speed reading is a reality or an illusion.)

We then can try to break down the role of affective influences, which must be studied in relation to input and storage and all the intermediate processing, coding, and final symbolic sampling and representation. We cannot allow ourselves to make any hasty generalizations about this. We know that there are children who perform better in examinations (better recall?) when they are frightened, whereas others perform worse and some who study better when they are alone and some who study better in crowds. This is related to the old problem of whether and to what extent a falling man or a drowning man actually reviews his life. Is this an experiment in hypermnesis or an illusory phenomenon?

As I have indicated above, it is impossible to conceive of retrieval without pathways along which a preconscious search can proceed. The richness and abundance of such pathways indeed may be the essential determining factor in retrievability. This too has never been studied in these terms. Nor has the influence of brain damage, drugs, or affective processes been studied in relation to them.

Another element is of critical importance: the element of remembering that consists not merely of recalling something in an almost impersonal and detached fashion but the form of recovery that is almost as if the original events were occurring again in the here and now. That this varies in degree of vividness and immediacy is clear (Fisher, 1956). Here again temporal variables come into consideration. These are important not only for the understanding of remembering itself but also because this aspect of the process of remembering has importance for many aspects of psychological health and illness. The vividness of reliving can have effects on the clarity and security of the boundaries between reality and fantasy, between delusion and dream, and on the phenomena of depersonalization and derealization. Moreover, it is intimately linked to the autonomic or affective or limbic or what I have called the "gut" component in remembering.

It also is related to creativity and to the learning process, which like remembering itself is predominantly a preconscious process up to the end point at which symbolic sampling occurs, with its inevitable feedback from the symbolic sample to the ongoing preconscious stream. This has been studied in some measure under hypnosis but little if at all in other ways.

That the complexities I have spelled out may arouse resentment and impatience is natural enough. But unfortunately we cannot make things simple, either by wishing or by fulminating. We can render them simple only by working with their complexities until we find in them a simplified pattern. Premature simplification dooms us to failure. Perhaps, since I have tried in some measure to work on both sides of this fence, I have some right to repeat my statement that one function of the experimentalist, anatomist, and pathologist is to remind the clinician of the need for precision in observation, in records, and ultimately in quantitative appraisals, while on the other hand the function of the clinician toward the experimentalist, pathologist, and anatomist is to remind them of the full complexity of things as they occur in nature, so that they will not oversimplify. Research work and mathematical models always require the artifice of simplification. The mere effort to limit and reduce the number of variables is a simplifying artifice—a justifiable one but one we must never mistake for the full reality. So the clinician serves as the experimentalist's conscience just as the investigator, anatomist, and pathologist serve as the conscience of the clinician.

To this I should add that human beings are not notorious for liking their consciences, so it is not remarkable that these partners do not always feel or act like the best of friends. Yet if we face the fact that each of us serves a basic and necessary function for the other, maybe we can forgive one another our differences and work together.

At least this hope seems to me to justify the really awesome tapestry I have tried to emphasize in my presentation.

IV. Summary

The following, then, are the issues which, in my opinion, demand investigation:

1. I place primary emphasis on what I call "preconscious processing" as the core of all thinking and of all retrieval of past experience.
2. I point out the importance of preconscious input and also of preconscious somato-muscular and autonomic responses as preparations for symbolic representation.
3. I emphasize the importance of an ingredient in remembering that usually is overlooked, namely, the variable capacity to reactivate past experiences in a form so vivid as to constitute a partial reliving of the original experiences, including their affective or "gut" components.
4. I call attention to the importance of determining just what is meant by "hypermnesis" and whether it is a fact or an illusion, since if it exists as a real phenomenon we cannot hope to understand reductions in remembering without parallel study of its enhancement. Except for some studies of its special manifestations under hypnosis and a few studies of the "idiot savant," studies of the contrasting factors that determine hypermnesis have yet to be made.
5. I emphasize the importance of the fact that the process of remembering is an essential ingredient in the automatic establishing of ongoing reverberating processes.
6. Finally, I emphasize the fact that the studies of remembering and forgetting that are limited to the studies of the symbolic sample cannot be more than partial.

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MODALITIES OF MEMORY

Jerome S. Bruner

When Dr. Talland asked me to participate in this symposium, I told him that I did not really know anything about the psychopathology of memory, had never worked directly in the field, and indeed had worked little in general on memory. But during most of my research life, I have been concerned with processes that are highly dependent on what has been stored of one's past encounters with the environment.

So let me say a word about the bias with which I approach the problems of memory and how I approach the problems of keeping records of past encounters with the environment. Both of the fields in which I have worked extensively—perception and problem-solving—depend to an enormous degree on the retrieval of information relevant in particular contexts for the solution of a particular problem. In the last few years, working on infancy, I have been forced to become more conscious of this bias, as I shall presently relate. One begins with the question of the fate of the short-term memory trace, a problem worked on brilliantly in the last several years by a number of people—Melton, the Treismans, Colin Cherry, Broadbent, and many others. There emerges a fairly good, if rudimentary, picture of the kind of process that one may speak of as a reverberating echo or—as Ulric Neisser (1967) calls it—an icon that develops and persists until recoded in some way. Unless recoding occurs, there is little if any persistence. Survival depends on some process of consolidation. At the level of short-term memory, many phenomena obey George Miller's law of "the Magic Number Seven" (1956), a law that weakens as the time span of memory increases. How does short-term reverberation, if I may call it that, get transformed so that it affects the long-term behavior of an organism afterwards? How is this recoding or reconsolidation of the short-term trace effected? Surely not just by the modality of recoverable, specific event memory.

Let me begin, then, by asking what we mean by memory. I think, first of all, that we have to make a very sharp distinction for analytic purposes between the two great divisions of memory—what may be called *memory with record*, where specific events are recoverable, and *memory without record*, in which encounters are converted into some process that changes the nature of an organism, changes his skills, or changes the rules by which he operates but which are virtually inaccessible in memory as specific encounters. The person who learns to play tennis with great expertness cannot give you the specific set of instances that led to the perfection of an aggressive backhand. Yet you would have to say that this kind of skilled behavior is a retention of past encounters just as genuinely as is the player's memory of having served a particular boomer on one particular occasion at the Longwood Cricket Club. It reflects the past in a different way.

Why do certain past encounters get carried with a tag of specificity while others get converted instead to skills or rules or generic modes? They clearly must be quite different neural mechanisms, as we know from neuropathology. Apraxias and amnesias are not much akin. Corkin (1968) records a case with highly impaired memory capacity who shows virtually unimpaired ability to master skilled motor tasks; she cites comparable cases in the literature.

I. The Storing of Information in a Skill

A skill is a mode of sensory-motor functioning that provides rules for anticipating and responding to categorized situations of varying uncertainty. The rules appear to be organized in a fashion not easily described. It is as if, to borrow an image from the logician Bar-Hillel (1966), our skilled knowledge was stored in a set of encyclopedic volumes that were organized not alphabetically but rather from left to right in the order from generic to specific. In any given instance, given a task requiring skilled sensory-motor coordination, we enter the volumes at the left at the most generic level and deal with the problem in terms of relevant solutions found there, only going on to the right as needed. The generic skills include, for example, such primitive ones as centering the body with respect to an object to be reached for, distributing the weight of the body between the two legs before exerting force, etc. It is only as we move from left to right in these volumes that we get anything at all approximating specific "record" memory—how to deal with a *particular* contingency encountered under *specific* circumstances and dealt with in a relatively unique way. Interestingly enough, skill (particularly when looked at in this way) is notably transferable and, in the linguist's sense, highly generative.

It will come as no surprise to you that such a model of how information is stored in skills was thrust upon me by studies of the ontogenesis of human skills. It is, I think, an extension of the models of differentiation in growth long ago proposed by Coghill (1929), Herrick (1924), and others. Perhaps it would be best to look at some specific examples.

Consider some observations on infants of 4-8 months "learning" the skills of hand-mouth and hand-eye coordination. Typically, in the earliest period of reaching the child passes through a stage of relatively undifferentiated activity, involving as much activation as directional orientation. We present a "hypertoy" in the form of a bright red ball with white concentric rings, outlined with black edges, with a low-reflecting black velvet patch at the center, surmounted by a pearl—bright, concentric, contrastive, and generating much binocular parallax as it rotates and moves slightly with respect to the infant's line of regard. The child looks fixedly, lines up on the object, and shows a phenomenon of "pumping up" or activation that involves the tonic innervation of arms and trunk that bring the arms into a "ready" position. We infer that there is as much by way of mobilization of intention as of action, because it is often the case with the 3½- and 4-monther that once the ready posture and sufficient activation is achieved, the hand may swipe out in the direction of the ball, a kind of ballistic movement. If the infant retrieves the object, it is almost invariably brought to the mouth. It is difficult in this sequence of behavior to differentiate segments in the behavior observed. It seems not to be constructed of modules that go off, with pause for adjustive feedback between. Its sequential character, rather, is diffusely organized, and when one observes the behavior in frame-by-frame cinematic analysis, one is struck by the uncertainty with which the intention is executed.

By the fifth or sixth month, the child not only reaches more efficiently, but certain other things are evident. Instead of the object always ending at the mouth, once caught it becomes free of a terminal point.

It can be mouthed or banged or dropped. The skill itself begins to develop modules. The child reaches out, pauses, closes in, pauses, changes direction, grasps, and so on. Since the mouth is no longer the sole terminal point, the modularization of certain segments of the skill make recombination possible. The child takes the ball toward his mouth, mouths it, takes it out and looks at it, puts it back to the mouth, and indeed even drops it and retrieves it. It is as if the modularization of the activity makes it possible not only that he is now more skillful at being able to generate action sentences, so to speak, he can also recombine modules of an act in such a fashion as to generate much more skilled activity than one would predict just from knowing what it was that he had learned. He has learned not only a skill but also certain rules for transforming that skill to deal with new situations.

There also appears to be learning to anticipate in a fashion that does not reflect specifically any past event, or record even a straightforward summary of the past. Consider how the child shapes his hands to the requirement of that which he is reaching for. At the outset, as noted, the child "flings" or "swipes" his hand toward the object. Obviously, there is much anticipation in this activity, because there is no evidence of surprise when the infant connects, and the aim of the ballistic swiping is extraordinarily good. As my colleagues Trevarthen and Richards (1968) quite properly insist, there must be a considerable amount of preadaptation in order for this line of ontogenetic development to recur at all. But the infant may reach the object with a balled fist and not be able to take it. Gradually he develops a wide open grasp —the hand with fingers spread out like a bunch of bananas—maintaining this open hand until such time as the object is touched, when it closes in. Now the corollary discharge of intention has achieved a new sequential appropriateness. It now becomes possible for a new skill to develop. By 8 months the shape of the reaching hand takes on the shape of the object toward which it is reaching.

Now let me make just one other observation before I leave the skill area. Piaget (1954) reported some years ago that the child's memory for a hidden object takes a motoric form. The observation has been repeated many times. A child is playing with a rattle, shaking it. He moves his hand to the edge of the crib. The rattle drops. The child then shakes his hand and looks at his hand—no rattle. He shakes his hand again—no rattle. When the experimenter approaches the crib, the infant will again start reaching out, wanting the rattle. The motoric behavior is in effect "carrying" memory.

The difference between my first examples—where past encounters are converted into generic skills as the infant develops—and the example from Piaget is in specificity, but not in mode. In each case the past is represented enactively, in a sensory-motor organization. But in the one, it is the memory of a concrete event that is involved and in the other some general sensory-motor rule. I rather suspect that it is the enactive representations of specific instances that become converted into generative skills of the kind we were considering. How this occurs is only poorly understood, although there are some very interesting suggestions to be found in the recently translated book of the late Russian physiologist Nicolai Bernstein (1967). I would like to suggest, simply, that the conversion comes in the effort to develop means of reducing discrepancy between what Bernstein calls the *Sollwert* and the *Istwert*—sensory-motor ways of keeping the difference between the two at a workable level. This involves a reduction in degrees of freedom as a first means (as when the infant prevents premature closure of the fingers during reaching by holding the hand rigidly wide open). It is this reduction in complexity that, for a guess, begins the process of stripping instances of their uniquely memorable properties and converting them into the elements on which induction can be established.

Now let me come to another type of storage without record. I refer to the formation of a spatio-temporal schema—one's working image of a room, a city, or a cockpit. Successive encounters are summed up or simultanized into such schemata. They are highly selective schemata: like the New Englander's image of the United States—a huge Cape Cod with a third of the United States between Boston and Dedham, the rest undifferentiated to the west. Patterns of this type probably govern and help integrate the world of vision and the world of touch. Consider some examples from infancy. We have studied how an infant learns to use a cup. If a cup is a trifle heavy, it may lag a little bit in being lifted. The free hand that is going to meet the cup at the frontal midline may be higher than the cup held by the other hand. The 7-month child will at this point close his eyes while he fishes in his kinesthetic space to get hold of the cup and then will open his eyes to reconfirm. He only gradually gives this up. It is not that there is initially some disconnection between the spatially extended field and the motoric field in which he operates but rather a question of learning to cope with the anomalies of space and motor activity. Gradually the infant builds a schema of a sensory-motor space that he can rely on. There is an old question: "Does the eye lead the hand or the hand lead the eye?" Perhaps the real issue is "Does the eye mislead the hand or does the hand mislead the eye?" The eye more frequently misleads the hand, and so the eye may get momentarily shut out until such time as there can be a trustworthy kinesthetic organization of space. What is happening is that the anomalies, the special cases, and the hard tasks are getting summarized in working schemata.

Take another phenomenon. We have been doing studies of detour reaching, getting around obstacles with the hand. We find, for example, that when there is a screen across half of the visual field and an object is presented first in the open part of the field, the child initially reaches for the object with the hand ipsilateral to the object. We now place the object behind the screen. The 8-month-old child will reach with the ipsilateral hand again but quickly get distracted by the screen itself. At the next stage typically the child will start reaching again with the hand ipsilateral to the screen, and the motoric component takes over. The hand goes to the edge of the screen and doubles back behind the screen to get the object while the other hand (within inches of the back of the object) remains unoccupied on its side of the midline. Usually by 14 months the child begins to appreciate an organization of the spatial field independent of an action initiated within it. It becomes possible for him, even if he starts with the hand ipsilateral to the screen, to stop and shift and reach directly behind with the contralateral hand and get the object without doubling back. Space is now something *in which* action occurs. It is not bound by action.

Professor Piaget (1954) has discussed similar phenomena as "decentration"—being able to appreciate the structure of a situation independently of one's position within it. It is an important concept, I believe. I would want to broaden it to the notion of "objectification," appreciation of the existence of constraints in the environment that are in the environment, not in one's actions on the environment. Interestingly enough, decentration and objectification may require loss of specific memory tagging in order to be effective. Memory tagging may be one of the factors that keeps one's view of the environment egocentric and subjective.

II. Memory Storage in Rules

Another type of storage without record that I would like to talk about is the kind that Heinz von Foerster discussed at a recent memory symposium presided over by Dr. Pribram. It is storage in the form of a rule. Encounters are converted into instances, from which a rule is induced—what von Foerster referred to as inductive inference. Von Foerster notes that if you wanted to store information in the form of a multiplication table for all digits up to 10^{10} by 10^{10}, it would take a book a billion miles, or one light year, thick. To retrieve a product would require on the average about a half light year. So nature has very benignly given us a technique for much more rapid retrieving. We buy a calculator, a prosthetic device, built according to a rule. But we learn not only a rule (multiplication) but also a procedure. The rules of procedure usually involve strategies that permit extraction of general information through throwing away specifics. Indeed, all abstraction procedures are devised precisely to *legitimize* the forgetting or ignoring of specific, rich information.

Now we may return to the question of memory proper: memory of dates, names, places, sights, sounds, and smells. We store skills, rules, and schemata. To this there is added the marvelous frosting that permits us to remember mother, father, friends, Fiesole, or the scent of a certain ball gown. But I urge we remember that this is a highly specialized form of storing the past and not necessarily amenable to study in isolation from the others. We do not regard it as pathological if the scientist has no recallable memory of instances on which a generalization is based. We might well ask about pathology in memory—whether an amnesia shows any inability on the part of the patient to use or to think in terms of the *type* of information involved in the amnesia or to great imagery related to it. Amnesia, apraxia, and agnosia, we must bear in mind, are quite as artificially separated as memory, action, and perception.

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ABSTRACT AND PICTURE-STRIP

Arthur Koestler

In Stevenson's novel *Kidnapped*, Alan Breck makes the casual remark: "I have a grand memory for forgetting, David." He speaks for all of us, and I do not mean only those of us afflicted with aphasia or senility. As a person who has indulged in several volumes of autobiography, I am painfully aware that *most* of my memories are like the dregs in the wineglass, the dehydrated sediments of perceptions whose flavor has gone, or, to change the metaphor, they are the dusty abstracts of past events on the shelves of a dimly lit archive—most, but not all, because this is only half the story. The other half is our capacity to recall scenes, or fragments of scenes, with almost hallucinatory vividness. The first could be called the *abstractive* type of memory, and the second, the "vivid fragment" or "picture-strip" type of memory. Elsewhere (Koestler, 1964) I have ventured the suggestion that abstractive memory and picture-strip memory are based on two different mechanisms, although of course they interact all the time. I am now going to stick out my neck in defense of this speculative hypothesis.

Take abstractive memory first. The bulk of what we can remember of our life history, and of the knowledge we have acquired in its course, is of the abstractive type. It must have a hierarchic structure like a thesaurus or a library subject catalog, with headings and subheadings as nodes on the branching tree. If one looks at a phonograph record with a magnifying glass, one is tempted to ask the naïve question: Why does the nervous system not produce engrams by this simple method of coding, instead of being so complicated? The answer is, of course, that a linear engram of this kind would be ideal for the reproduction of nonsense syllables learned by rote but completely useless for analyzing, matching, and recognizing input patterns by similarity and cross reference. The library must have a hierarchic structure. But the library is also subject to the laws of diminishing returns. Quite apart from the countless volumes that are left to rot away or fall to dust, there is a hierarchy of librarians at work who ruthlessly condense long texts into short abstracts and then make abstracts of the abstracts. I watch a television play. The exact words of each actor are forgotten by the time he speaks his next line and only their meaning remains. The next morning I can remember only the sequence of scenes that constituted the story. After a month all I remember is that it was a play about a gangster on the run. Much the same happens with the mnemic residues of books one has read and episodes one has lived through. The original experience has been stripped, skeletonized, reduced to a colorless, schematic outline. This process of sifting and abstracting starts long before a particular input is put into the memory store. Perception and memory-formation are continuous processes; at every level of the perceptual hierarchy through which the input traffic must pass on its ascent from periphery to center, it is analyzed, classified, and stripped of all irrelevant detail. To start with, there is lateral inhibition, habituation, and presumably some efferent control of receptors. On the higher levels are mechanisms responsible for the visual and acoustic constancy phenomena, the scanning and filtering devices that account for the recognition of patterns in space and time, and enable us to abstract universals and discard particulars. The colloquial complaint "I have a memory like a sieve" may be derived from an intuitive grasp of these filtering devices that operate all along the input channels and the storage channels.

The word "abstract" has, in common usage, two main connotations: It is the opposite of "concrete" in the sense that it refers to a general concept rather than a particular instance; in the second place, an "abstract" is a summary or condensation of the essence of a longer document. The end result of all this filtering is the dusty abstracts in the memory store into which Tolstoy's collected works have shrunk.

This impoverishment of lived experience is unavoidable; the processes of generalization and abstraction imply by definition the sacrifice of particulars. But this, of course, is not the full story; there are several compensating mechanisms. In the first place, memory is not based on a single abstractive hierarchy but on a multitude of interlocking hierarchies, pertaining to different sense modalities such as vision, smell and hearing. What is less obvious is that there may exist several distinct hierarchies with different criteria of relevance operating within the same sense modality. I can recognize a melody regardless of the instrument on which it is played; and I can recognize the timbre of an instrument regardless of the melody played on it. One is thus led to assume that melody and timbre have been abstracted and stored independently by separate hierarchies within the same sense modality but with different criteria of relevance. One abstracts melody and treats everything else as noise; the other abstracts timbre and treats melody as noise. Thus not all the information discarded as irrelevant by one filtering system is irretrievably lost, because it may have been stored by another filtering hierarchy with different criteria of relevance. Recall then would be made possible by the cooperation of several interlocking hierarchies, which may include different sense modalities or different branches of the same sense modality. You may remember the words of the aria "Your Tiny Hand Is Frozen" without the tune. You may remember the tune after having forgotten the words. And you may recognize the sound of Caruso's voice on a phonograph record, regardless of the words and the tune he is singing. Recall, then, could be compared to the process of multicolored printing by the superimposition of several color blocks. Some modest evidence for this hypothesis can be found in a series of experiments by J. Jenkins and myself (Koestler & Jenkins, 1965; see appendix), and more tests on these lines can be designed without much difficulty. Needless to say, recall will be the more complete, the more branches of the perceptual hierarchy have participated in the process of analyzing and storing an experience.

All this refers to abstractive memory. Now to the second type, the "picture-strip" type of memory. It is a method of retention based on principles that seem to be the exact opposite of memory formation in abstractive hierarchies. It is characterized by the recall of scenes or details with almost hallucinatory clarity. They are like photographic closeups, in contrast to abstractive memory's aerial panoramas seen through a haze. The emphasis is on the detail, which may be a fragment torn from its context that survived the decay of the whole to which it once belonged, like the single lock of hair on the shriveled Egyptian mummy. It may be auditory—a line from an otherwise forgotten poem—or visual—a wart on Nanny's face—or a taste or smell. Though often trivial from a rational point of view, these fragmentary images may have an uncanny evocative power. We thus are led to assume that while redundant by logical criteria of relevance, they have some special *emotive* significance (on a conscious or unconscious level) that caused them to be retained for permanent storage. Nobody, not even computer designers, think all the time in terms of abstractive hierarchies; emotions color most of our perceptions, and there are indications that our emotional responses also involve a hierarchy of levels – including midbrain structures and archicortex, which are phylogenetically older than the structures concerned with abstract conceptualizations. One might speculate that in the formation of "picture-strip" memories these older levels in the hierarchy play a dominant part.

There are some further considerations in favor of such a hypothesis. First, from the neurophysiologist's point of view, they receive strong support from the Papez-MacLean (MacLean, 1961) theory of emotions. Second, from the standpoint of the communications theorist, abstractive memory generalizes and schematizes, while the picture strip particularizes and concretizes – which is a much more primitive method of storing information. Third, from the standpoint of the psychologist, abstractive memory would be related to insightful learning and picture-strip memory to a process resembling imprinting. But imprinting in Konrad Lorenz's geese is restricted to a critical period of a few hours and apparently results in a very coarse and vague imprint. On the human level, imprinting may take the form of eidetic imagery. According to Jaensch (1930) and Kluever (1933), a considerable proportion of children have the eidetic faculty – they are able to "project" a photographically accurate, colored image of a previously fixated picture onto a screen and to repeat this after long intervals, sometimes even years. Penfield and Roberts' (1959) experiments, evoking what is claimed to be the total recall of past scenes by stimulation of the patient's temporal lobes, may be a related phenomenon.

But though quite common in children, eidetic memory tends to vanish with the onset of puberty, and is rare among adults. Children and so-called primitives live in a world of visual imagery, and languages like the Bushman's have been described by Kretschmer (1934) as "picture-strip languages." In William Golding's novel *The Inheritors* (1955), the author has his Neanderthalers say, instead of "I thought of something," "I have a picture in my head" – and for once a novelist seems to have got the picture right. The eidetic child's way of "imprinting" pictures on the mind may represent a phylogenetically and ontogenetically earlier form of memory formation – which is lost when abstractive, conceptual thinking becomes dominant.

To sum up, abstractive memory, operating through multiple interlocking hierarchies, strips down the input to bare essentials according to each hierarchy's criteria of relevance. *Recalling* the experience requires dressing it up again. This is made possible, up to a point, by the cooperation of the hierarchies concerned, each of which contributes those aspects it has deemed worth preserving. Added to this are touches of "vivid detail," perhaps fragments of eidetic imagery, which carry a strong emotive charge—and the result is a kind of collage, with glass eyes and a strand of genuine hair stuck on to the hazy, schematized picture.

Appendix: Inversion Effects in the Tachistoscopic Perception of Number Sequences[1,2]

Arthur Koestler and James J. Jenkins
CENTER FOR ADVANCED STUDY IN THE BEHAVIORAL SCIENCES

ABSTRACT

Experience suggests that a common error in processing visual sequences is inversion or transposition of two or more adjacent items. This phenomenon suggests that information concerning the identity of items and their positions may be partially separable. A perception experiment was performed with tachistoscopic exposure of 5, 6, and 7-digit sequences. Abundant evidence was found for transposition errors. Further, such errors were distributed in a serial position curve much like that found for errors of single items.

PROBLEM

While information-processing in visual perception has received increasing attention in recent years (e.g., Sperling, 1960; Averbach, 1963; Broadbent, 1963) one common phenomenon of faulty processing which may have some theoretical significance seems to have been

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[1] The writers are indebted to Donald Foss for collecting and coding the data. Thanks are also due to Professor Douglas Lawrence and Professor Ernest Hilgard of Stanford University and to Professor Arnold Mechanic and Joanne D'Andrea of California State College at Hayward for their generous facilitation of the study.

[2] Reprinted with permission from *Psychon. Sci.*, 1965, 3, 75-76.

ignored. We refer to the inversion (or transposition) of adjacent items in a sequence of numbers shown in a tachistoscope. Though such errors are common enough in bookkeeping and have earned a special proofreader's mark, they are absent from discussions of visual perception or memory span in standard works such as Osgood (1953) and Woodworth and Schlosberg (1954).

Apprehending a series of numerals and subsequently repeating them in their correct sequence must either involve the *ordered* storage of the individual items or the storage of information relating to that order. Both information identifying an item and information defining its place in the sequence must be available for the S for successful performance of the task.

The potential separability of the two kinds of information involved is not easy to demonstrate. If a subject makes a single error of identity, reporting either an incorrect number or a blank, it may indicate that he has lost only identity information. This argument, however, is inconclusive, because if the subject had acquired no information at all regarding the offending item, but complete information regarding other items, the outcome would be the same. The inversion of two digits or the permutation of three or more digits, on the other hand, furnishes a compelling argument because it is *prima facie* evidence that the identity information is accurate while the positional information is incomplete or distorted.

The purposes of the present study were to demonstrate that the phenomenon of transposition could be observed under laboratory conditions and to describe the locus of its probable occurrences in a given sequence.

METHOD

The stimulus materials were 80 4 × 6 notecards upon which digit sequences were typed in elite type. The 80 sequences were divided into four sets of 20 cards each. The first set showed sequences five digits in length; the second and third sets showed six-digit sequences; the fourth set showed seven-digit sequences. The sequences contained the digits 1-9 with never more than a single digit repeated on a given card. The repeated digit, if any, never occurred without at least one intervening digit. The sets were presented in the order given above. A random arrangement was made of each set. This arrangement was used in the forward order for half the Ss and in reverse order for the remainder. The materials were presented in a mirror-type tachistoscope.

The *S*s were 14 undergraduates in introductory psychology courses. The *S* held a plunger switch which activated the tachistoscope. The *E* gave a ready signal when the stimulus card was in place. The *S* activated the tachistoscope when he was ready. He was instructed to say the digit sequence aloud immediately after its appearance, and was encouraged to guess if he was not sure of one or several items. The *S* always knew how many digits were shown. Responses were recorded on a tape recorder. Only one exposure per sequence was given and the *S* was not given any information about the correctness of his response.

Two practice sequences with ascending limits were given to accustom the *S* to the apparatus and to provide the *E* with some information on threshold. The test sets were then presented. One-minute rest periods were given after each set.

Exposure duration was individually adjusted for each *S*. Pilot work suggested that transpositions occurred most readily at the point where the *S* was beginning to miss single digits in the sequence. Therefore, the *E* attempted to have the exposure interval long enough that the proper number of digits would be reported but short enought that they were not always reported with complete accuracy. After every five cards the *E* decided whether to keep the exposure the same or to change it. Since there were practice effects in the task and since the task became appreciably more difficult, *E* continued to modify the presentation time during the course of the experiment. Generally 10 msec steps were employed in such changes but with an occasional *S* whose performance was markedly inferior the step span was increased.

RESULTS AND DISCUSSION

Responses were transcribed from the tape and scored. The following categories were employed:

C —correct
E —gross error
I —one digit incorrect, or "blank" reported for a single missing digit
T —transposition of adjacent pairs of digits with rest of sequence correct
T′ —transposition of three or more digits with remainder correct
IT —transposition of two or more digits *and* one digit incorrect
O —other errors, usually experimental or equipment errors

Results are given in terms of these scoring categories in Table I. Ex-

Table I
Distribution of Responses by Categories
for Each Stimulus Set (280 items)

Scoring code	5 Digits	6 Digits	6 Digits	7 Digits
C	130	60	65	12
E	21	67	47	122
I	50	43	50	21
T	23	23	32	12
T'	2	14	5	9
IT	44	64	73	96
O	10	9	8	8

amination of the table shows that transposition provides an important source of errors. It is, however, difficult to find a statistical model which would provide a precise evaluation of the statistical significance of such errors. As Woodworth and Schlosberg (1954, p. 697) point out in their discussion of scoring memory span, any scoring system which attempts to provide separate credit for accuracy and order is arbitrary. Thus, any statistical model must make assumptions about the *S*'s strategies on the one hand (e.g., Did the *S* note that digits can repeat within a sequence and, if so, did this alter his guessing behavior in the appropriate manner?) and the interrelationships of error types (which we do not yet know) on the other. Fortunately, the question is not crucial for present purposes. The only question that need be asked here is whether there is more transposition than would be expected by chance (however chance is to be defined).

We think the answer to that is clear. Of the 140 errors on the five-digit sequences, 69 involve transpositions; of the 211 and 207 errors on the six-digit sequences, 101 and 110 respectively contain transpositions; of the 260 errors on the seven-digit set, 117 contain a transposition. It is evident that until the task becomes exceedingly difficult (and the response unscorable), approximately half the errors involve transpositions. No reasonable "guessing" or chance model we have contrived can account for this finding. It seems simplest to conclude that in a large portion of the errors the *S* has the correct information as to the identity of some of the digits but has lost the information as to their precise location.

As a first step in describing the phenomenon the distribution of errors over positions was obtained for the simplest errors of both types. Table II shows the location of the error for each instance when one digit was incorrect (I error). Table III gives the location of the pair of items transposed when only single transposition was observed (T

Table II
Position of Errors in Cases of a Single Incorrect Digit

Sequence	Position						
	1	2	3	4	5	6	7
5-digit	1	1	4	34	10	–	–
6-digit	0	2	1	4	31	6	–
6-digit	0	1	3	11	25	10	–
7-digit	0	0	2	0	7	8	4

Table III
Position of Transposed Digits in Errors Involving a Single Transposition

Sequence	Positions transposed					
	1-2	2-3	3-4	4-5	5-6	6-7
5-digit	0	2	18	3	–	–
6-digit	0	2	1	17	3	–
6-digit	0	5	1	21	5	–
7-digit	0	0	0	3	7	2

error). It can be seen that both sets of distributions for all sequence lengths show the same serial position effect, suggesting that both kinds of error are susceptible to the same form of interference. If one has all the individual items, he is least likely to have precise position information in the latter half of the sequence. Conversely, if one lacks the identity of an item, it is most likely to be one from a position in the latter half of the list. The most probable transposition for any particular length of sequence appears to involve the inversion in order of the item in the most difficult position in the sequence and the item immediately preceding it.

The psychological nature of each kind of error is not at all clear but it seems likely that future work will help narrow the alternatives. It would be particularly interesting to know, for example, whether transposition is equally common when the experiment is conducted with Sperling's procedure or with a rapid sequential procedure such as that used in short-term memory research.

While we cannot at present make any decision as to the underlying nature of the transposition phenomenon, we feel that this experiment concurs with common experience in pointing to a pervasive distortion in the visual perception and reporting system which theories of information processing must take into account.

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IV

CONCLUDING REMARKS

INTERACTION BETWEEN CLINICAL AND LABORATORY RESEARCH ON MEMORY

George A. Talland

Trade between experimental and clinical students of memory has been unbalanced at least from the beginning of this century. Ever since Kraepelin, German neuropsychiatrists have turned with confidence to experimental psychology for techniques to verify and quantify the inescapable, albeit at times complicated, evidence of their clinical observations and to fit these into a coherent theory. Ebbinghaus and G. E. Müller furnished the first standard methods for the investigation of memory, and subsequently the repertory expanded under the influence of the Gestalt school. Elsewhere concepts and techniques derived from Pavlov have been used widely for several decades. In exchange, there have been the experiments on the influence of affective tone upon retention, derived somewhat tenuously from Freud's construct of repression, and above all Bartlett's (1932) indebtedness to Head for the concept of the schema.

The recent revival of interest in memory, as distinct from learning, among experimental psychologists has further tilted the scales, since every psychologist who studies patients with memory defects tests them on one version or another of the immediate recall span. We keep on rediscovering that men and women who barely remember an important incident that occurred last week can perform within normal limits on the standard digit span, and we draw the obvious conclusion that this technique is not of much use as a test of memory dysfunction.

Is the immediate retention span for unconnected items, in fact, at all relevant to normal memory function? The human capacity probed by this type of test is one aspect of attention—the amount of information a person can transmit with minimal recoding or organization. This is a capacity that indeed may set limits to performance both at the stage of acquisition and in the process of recall, although it is unusual for us to take in information or extract it from memory in discrete and unconnected units, and in virtual isolation from all other memories, as we are forced to do in tests of the digit span.

Patients with severe memory disorders do not perform much worse than healthy and normal people on these tests, because the task effectively reduces the latter to a level at which those patients can function. There is also a second reason why the span of immediate recall may be unrepresentative of remembering in general: It follows the presentation of the material without delay or any intervening event. It has been pointed out that if there are only two items to be recalled, the reproduction of the first does not follow immediately upon its presentation, since the second item intervenes between the two events. That, of course, is true, and moreover the enumeration of the earlier items of a string in the process of recall always creates some interference with the retention of the subsequent ones; but, if nothing else, experiments with amnesic patients have shown that this interference is very different in its effect from the interference of diverting activities.

As long as they can maintain a set directed to a particular task, amnesic patients are quite capable of following instructions, of recognizing the pattern of a line drawing, and of holding and reproducing meaningful sentences and some, if not all, of the items strung together arbitrarily into a list. Their chances of remembering what to do or say drop sharply, however, if their attention is diverted no matter how briefly. There are any number of clinical observations as well as some experimental findings that illustrate the inability of amnesic patients to pick up a thread after an interruption or some interpolated activity. If the same span of time is spent inactively, however, they are quite capable of resuming where they had left off. I believe they can do this, not thanks to subvocal rehearsal, but because nothing enters their minds during the unoccupied interval, so that subjectively there is no break in their performance. But this is a debatable proposition, and I have no convincing evidence to support it.

Tests of immediate recall do show up a deficit in amnesic patients if they involve some reorganization of the message or if the input exceeds the span of apprehension, as is the case with the running digit span. The processing of such material creates enough interference to disrupt the operations of registration or retrieval in amnesic patients, even though it may have no deleterious effect on healthy people, but it does not disrupt the subject's task-set—neither do the customary tests of delayed recall, with retention periods of 2–3 hr. I consider these as tests of short-term memory, although experimental psychologists usually regard them as tests of long-term retention and treat them as if they were representative of the operations involved in the recall of old memories. That they are not is not only because no experimenter can squeeze into a laboratory session the equivalent of 25 years' worth of experiences but also because the circumstances are most conducive for the subject to retain and resuscitate at the time of recall the task-set he adopted at the time of learning.

I am quite ready to agree with Bartlett that remembering involves the restoration of the attitude brought to the original experience, but alas, I cannot form any clear image of how this is done. Perhaps we might begin to clarify this notion by some parametric exploration along the pattern set by Tinbergen (1953) and by using as our independent variable some such thing as the two stones placed at different levels outside the front door of the Guermantes mansion, which—as you will recall—quite unexpectedly reminded Proust (1928) of Venice, through the sensation he "had once felt as he stood on two uneven flagstones in the baptistry of St. Mark's, and with that sensation came all the others connected with that day, which had been waiting in their proper place in the series of forgotten days, until a sudden happening had imperiously commanded them to come forth."

We could adjust the levels of the stones by degrees of just noticeable differences and apply some objective scoring system to our subject's responses at each grade, but I fear that these responses, however enlightening about other psychological processes, would not teach us much about the recall of old memories. No wonder that psychologists have not resorted to this technique. Indeed, the only instance of its application that I have come across recently is in the novelist Nabokov's (1966) autobiography. Some years ago he revisited Cambridge, where he had spent a couple of years as an undergraduate, and went to see his one-time tutor who completely failed to recognize him. Only when the novelist stumbled over a tray that was lying on the floor and upset a teapot did the tutor show a sudden flash of recognition, for the visitor thus reenacted the very accident that had marked his first visit as a freshman to the same room.

Examples of similar incidents are familiar to all of us, but they are strikingly absent from our encounters with amnesic patients. Those patients may recognize you but mistake you for someone else; they cast persons into unfamiliar roles and place true events in the wrong contextual setting. These errors may have some features in common with intraserial transpositions in ordered recall, but I doubt that an analysis of the latter will ever get, for example, at the root of so-called confabulations. Probably nobody seriously proposes that it will, but a distinguished experimental psychologist who is a leading figure in the current revival of "short-term memory" research has on record the opinion that "rote verbal learning is central to all human learning." All the lessons I have learned from a study of disturbed memory function force me to disagree with that opinion. People with memory disorders fail in their search operations, in the reconstruction and piecing together of memories. They are, of course, apt to be quite unsuccessful at rote learning as well as at any other type of learning; but that which they retain from past learning consists chiefly of skills acquired in a rote fashion.

It is for this reason that I doubt whether we can learn much from animal experiments about the pathology of memory, as distinct from defects in new learning. Appetitive or aversive responses to experimentally established cues represent the kind of learning that is quite effectively retained by the patient who can barely remember one incident from the past 5 years and who one moment thinks of himself as an adolescent and in the next correctly reports that he has grandchildren but is quite likely to mistake their number and names. The amnesic patient may perform successfully on the tasks we set him in the experimental laboratory, even in some tasks probing memory and learning, but an hour later he will have no recollection of those tests. Yet savings scores are quite likely to show some retention. This dissociation between performance and its registration for future recollection is not restricted to laboratory tests. Kohnstamm's (1917) amnesic patient preserved his skill at the piano and was capable even of learning new music. One evening he accompanied a singer in a concert held in the hospital, but the next morning he had no recollection of the occasion which, unquestionably, must have stood out experientially amid the routine of life on the ward.

Performance, the objective evidence that our behaviorist psychology relies on, often just skims the surface of a memory derangement and is apt to mislead us if it alone is admitted as evidence. It might just enable us to discover an anomaly in the amnesic patient who greets his doctor effusively when the latter returns after a few minutes' break in his examination, but we understand more of the patient's unexpected behavior if he also states his belief that he has not seen the doctor for days. Satisfaction with the first evidence of successful performance would not have revealed to Dr. Moyra Williams (Williams & Smith, 1954) that one of her patients, after recovery from an amnesic syndrome caused by tuberculous meningitis, still suffered from a considerable retrograde amnesia for the period that preceded his hospitalization. He could name everyone in a group photograph taken at a training course shortly before his illness, but he had no recollection whatever of the occasion that had brought him together with those men. Neither would I have discovered an unusual anomaly in recall if I had been content with the correct responses an amnesic patient gave to my questions probing his memory for some events that had happened prior to, and others that had taken place since, the onset of his illness.

This young man, a patient of Dr. Raymond Adams', twice within 30 min accurately answered my questions and both times mistakenly believed that it was I who had just imparted all that information to him. Nor would the evidence of performance have justified Claparède's (1911) somewhat cruel experiment when he jabbed a patient's hand with a needle hidden between his fingers as he shook it greeting her. A few minutes later, as he once more extended his hand to her, she refused to take it. When asked to explain her refusal, the patient justified it by the fear that her hand might be jabbed but seemed to be completely unaware of the fact that this is precisely what had happened to her. All she could say in support of her fear was that people sometimes do hide pins between their fingers.

These examples—and others could be listed—illustrate the inadequacy of the concepts employed to evaluate acquisition and retention in verbal and other learning experiments for the analysis of pathological disorders of memory. Retroactive interference, to take one example, hardly offers a convincing explanation of forgetting in people who learn virtually nothing new that could compete with old learning; proactive interference is barely more convincing in view of their inability to recall much information acquired in the past and of the observation that the learning disability tends to correspond in severity to the extent of the retrograde amnesia. A deficit in proactive facilitation and response interference seem to hold more promising clues.

Experimental investigation of clinical memory disorders undoubtedly will continue to be designed so as to furnish parallels with the results of research conducted in the Ebbinghaus tradition and also with the type of learning that can be mastered by animals in which the neuropathology as well as behavior is amenable to laboratory control. These can be perfectly useful experiments, but in my opinion they should be supplemented with experimental research in the abstraction, coding, structuring, and sequential integration of information; in searching and matching strategies; in the planning, pursuit, and completion of hierarchical action plans; and in the ability to shift between alternatives, to correct errors in successive steps, and to utilize partial cues. Many of these experiments need not be concerned directly with memory function, that is, with the acquisition and retention of information. By setting a perceptual or motor task, they just as effectively could probe certain other basic mental functions that also are involved in new learning and in the voluntary retrieval of information.

The repertoire of the experimental psychologist includes several procedures that have been designed and developed to test some of these processes or closely related ones. A few of these techniques can be readily adapted to the level of patients with memory disorders: still others will have to be designed specially for them; and the most valuable should be those set up to test the accuracy of a clinical hunch. Other testing procedures will be dictated by a general theory, psychological or possibly neurological, and those too can make a valuable contribution to such an exploratory program in research, even though none of our current theories or models is comprehensive enough to accommodate within its framework all the phenomena observed in the pathology of memory.

The experimental laboratory has many valuable techniques and theoretical constructs to offer the clinical investigator of memory dysfunction, and these are not confined to the area of research marked as learning and remembering. This area itself, of course, is expanding and being enriched with concepts derived from external sources. The contribution from clinical research on memory disturbances, however, has not been very notable to date. I hope it will increase and that our symposium will have helped in promoting trade that way, because I doubt that the revival of research on memory in experimental psychology will accomplish much if it remains restricted to tests in short-term retention and to theoretical concepts that account for forgetting in those tests.

Remembering and forgetting are not all-or-none or even unidimensionally quantifiable processes. Observations made on patients with chronic or transient memory disorders display a variety of paramnesic as well as amnesic disturbances, while our laboratory tests typically provide only for the alternatives of complete success or failure. In some instances, the analysis of the performance record may admit certain types of partial recall or single out for special attention examples of sharpening, leveling, assimilation, or errors of reversal, intrusion, or acoustic confusion but hardly ever provides for a representative sample of the diverse errors that can occur in remembering. Even an inventory of the different types of faulty recall and recognition observed in life situations might not be exhaustive. I doubt that it would include an example of a person who correctly remembers certain events but later attributes their recall to his interviewer.

Systematic observations of psychopathological cases can provide clues to the processes involved in memory that could not be discovered by any other means. The discipline acquired in the experimental laboratory is an asset in making such observations, if only as a protection from a facile acceptance of venerable clinical theories. It also should help in translating casual observations or loosely formulated explanations into testable hypotheses. This may not always prove feasible, but it ought to serve as our goal if we wish to enrich academic psychology with material drawn from psychopathology and to formulate psychological models that fit the available models of brain function more closely than do most of those currently in use.

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